# THE JOURNAL OF PERIODONTOLOGY

*January*
1934

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COLUMBUS, OHIO.

*Published by*
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME V
NUMBER 1.

RKI
T355
v.5-8

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely, will make the Journal a fund of extensive information readily available to students, teachers, researchers, and clinicians.

This Journal is the official organ of the American Academy of Periodontology and will contain therefore, in the January issues, those papers of scientific and practical interest which are presented at the annual meetings of that society. This issue of the Journal contains the papers read at the twentieth annual meeting held in Chicago, Illinois, August 3-4-5, 1933. The July, 1934 number will contain the abstracts of articles on periodontia which appeared in the year 1933, complete.

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## Announcement

The Twenty-first Annual Meeting of the American Academy of Periodontology is to be held at the Minnesota Club in St. Paul, Minn., August 2, 3, 4, 1934. All members of the American Dental Association are invited to attend this meeting.

—Clayton H. Gracey, *Secretary.*

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THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED THIRTY-FOUR

# Diseases of the Mouth[1]

*By* FREDERICK W. MERRIFIELD, D.D.S., M.D.
Chicago, Illinois

DISEASE may occur in the mouth as in any other part of the body. Some of the diseases are peculiar to the oral tissues, some occur in association with and as part of the manifestation of disease elsewhere in the body. Instances of the latter are seen with skin lesions and following abdominal surgery in the form of infections of the parotid glands.

The function of the dentist does not end with the discovery of caries in teeth or of edentulous areas and the correction of these conditions, but includes the necessity of a consideration of all the tissues which make up the oral cavity and the contained structures. Important as are the purely mechanical procedures in dentistry, the pathological possibilities are vital.

Examinations of oral cavities are often conducted in a most perfunctory manner and filling operations and restorations are completed in mouths which show gross calcific inflammations and peridental lesions. These remarks are intended to suggest the ease with which disease, more or less serious, may be allowed to go unrecognized, or to be completely ignored.

No mouth should be examined without a full realization that disease may exist therein, perhaps threatening the life of the patient, and equally important, that infection may be transmitted to the operator with disastrous consequences. Scrutiny of the lips, mucous membranes, floor of the mouth and the tongue, should precede examination of the teeth.

Some of the local mouth affections are to be associated with the digestion or metabolism of the individual. Blair mentions the fact that the close relationship existing between diseases of the digestive system and oral disturbances may be explained by the fact that toxins absorbed from the stomach and intestines are partially excreted in the saliva.

Ulceration may be simple or serious, the diagnosis obvious or obscure. Ulcers are usually characteristic and present typical bases—edges—discharges and reactions upon adjacent tissues, local or distant. Microscopic examination of ulcer tissue is often necessary.

[1]Read at the twentieth annual meeting, American Academy of Periodontology, August 5, 1933, Chicago.

Ulcers occur on any part of the body including the mucous membranes. From a surgical standpoint ulcers are classified as:

1. of traumatic or non-specific pyogenic origin;
2. of specific origin;
3. malignant ulcers.

Faulty nutrition of a part as a result of anemia or congestion, as exemplified in arterio-sclerosis or varicosities may lead to ulcerations. General debility such as occurs in nephritis and diabetes predispose to ulceration. Parts which become anesthetic or are cut off from their trophic centers may become ulcerated because of the fact that repeated irritation or injury may not be appreciated. Examples of such are the corneal ulcers occurring after injury to the fifth nerve and the perforating ulcers of the foot in Tabes Dorsalis. Malignancies ulcerate because the growth enlarges and becomes susceptible to greater irritation and trauma, and particularly because the deeper layers of the skin or mucous membrane are replaced by tumor cells and the superficial epithelium is soon worn off.

Ulcers due to trauma or non-specific pyogenic infection may occur as the result of any form of surface irritation. These irritations may be chemical, thermal, mechanical or infective in origin.

Simple ulcers occur in the mouth as a result of irritations from decayed teeth, erupting teeth, particularly in cases where the deciduous roots are pushed out through the buccal gums in the molar region, too harsh usage of hard bristle tooth brushes, from clasps or margins of artificial dentures and other mechanical injuries. Drugs such as phenol, scalds and burns produce excoriations and resultant ulcers. These ulcers vary in severity and the more chronic forms may resemble and sometimes do degenerate into epitheliomata. The simple ulcer is ragged in outline with irregular edges which are not undermined, but surrounded by an inflammatory area. The base as a rule appears yellowish red in color and is covered by a moist mucoid slough. The ulcer may or may not be painful.

Ulcers due to the long continued irritation of trauma may become indurated and the edges inverted due to tissue proliferation, as witness the terrific injuries found in the mouths of patients who persist in wearing ill-fitting or poorly adjusted artificial dentures. In these cases the adjacent lymphatic glands may become enlarged. Injuries of this type frequently become malignant.

The simple ulcers tend to respond rapidly to adequate treatment, which includes removal of the cause and the use of soothing washes. Mildly astringent washes are often of value. Persistence of the ulceration and failure to clear up under simple treatment indicate the possibility of syphilis or malignant change.

Dyspeptic ulcers and herpes frequently appear in the mouth. The dyspeptic ulcers occur on gums, tongue or mucous membranes. They are small and very painful and may be present in the mouths of persons in good health and be recurrent. The ulcers are not associated with constitutional disturbance, but are

usually associated with dietary indiscretion. The history of a preceding vesicle, intense pain, tendency to occur in crops and the absence of trauma and the definite circular outline of the ulcer, make diagnosis easy. The treatment rests with dietary regulation, mild catharsis, astringent mouth washes and the use of a simple escharotic upon the ulcer.

Herpes may occur on any skin or mucous membrane surface. On the face and in the mouth the condition often accompanies digestive disturbance and acute infections, particularly of the respiratory tract. They occur in alcoholics and syphilitics, but at times there is no apparent cause. Regardless of the cause Blair regards herpes as a trophoneurosis.

In the mouth herpes may occur on the lips, cheeks, tongue, palate and pharynx in order of frequence. On the lips they occasion little discomfort, but within the mouth may be accompanied by decided pain, particularly during mastication. The vesicles occur in clusters and are often accompanied by fever. They may dry up leaving crusts, but if the vesicle bursts a pellicle remains which if detached leaves a small circular excavation surrounded by a zone of inflammation. Treatment is usually simple. Spirits of camphor applied to the vesicles will often dry them up. Carbolic acid or zinc sulphate applied to the ulcers will relieve pain and hasten healing. Compound tincture of benzoin is often effective.

Of the infective ulcers, i. e. those of specific origin, luetic lesions are probably among the most important from a dental viewpoint. Tuberculous ulcers are not common in the mouth and leprosy is seldom seen except in the large hospital clinics. Vincent's infections with the predominating B. Fusiformis and Spirochete are probably those most commonly seen. Syphilis due to infection with the Spirochaeta Pallida is of particular interest to dentists because the lesions may occur on the lips, tongue, tonsils or mucous membranes and are decidedly infective in the primary and secondary stages. It has been estimated that in three per cent of cases the primary lesions occur in these locations. Many dentists have become infected as a result of contact during dental operations. Ulcerations occur in any of the three stages.

The first evidence of syphilis is the chancre. It occurs two to four weeks after infection. The chief characteristics of a chancre are indurated base, sharply circumscribed borders, dense hardness resembling cartilage, and indolence. The diagnostic features are, the long period of incubation, persistence, non-inflammatory, indolent character, superficial ulcer, markedly indurated base, and absence of subjective symptoms.

First there is a red spot or papule developing in the course of a few days into a hard infiltration which is sharply defined, dull red in color, indolent and painless. As an ulcer forms, the edges become ragged and undermined. There is a unilateral adenopathy in which there often figures an initial "pilot gland" enlargement. Differential diagnosis is made from herpes with grouped vesicles which rupture, leaving painful excoriations, and from epithelioma, where the

border is hard, nodular and waxy, the base indurated. Microscopic examination of exudate by dark field illumination confirms diagnosis.

A young man presenting for dental care had an intractable "sore" on his lower lip. The lesion was about the size of a dime and suggested an eroded papular appearance. He also complained of a swelling in the neck on the same side. Serological tests were positive for syphilis.

In the secondary stage of syphilis mucous patches in the mouth are constant lesions. They occur as round or oval areas resulting from the erosion of luetic papules. They are sharply circumscribed and covered by a grayish pellicle in which spirochetes are present. They may occur on any part of the oral mucous membrane, usually on the commissures of the lips, on the cheeks or tonsils. Mucous patches are extremely infectious and the cause of many extragenital and innocently acquired syphilitic infections. The lesions are often mistaken for the lesions of acute ulcerous stomatitis or trench mouth.

A freshman medical student brought his fraternity brother to the office because the young man had complained of a sore throat and mouth. Examination showed many mucous patches and the Wasserman was four plus, much to the astonishment of the unfortunate boy.

Oral manifestation of tertiary syphilis is the gumma occurring as a nodular infiltrate in tongue, lip or palate. It is a circumscribed rather hard tumor which tends to break down and ulcerate and may be confused with malignancy or tuberculosis. Serological tests and microscopic examination are important in differentiation.

A case seen recently presented with complaint of a swelling in the mid-line of the tongue toward the base which interfered somewhat with articulation, rendering speech somewhat "thick". The swelling had progressed slowly and painlessly. A luetic history was obtained and the Wasserman test was positive.

Ulcerations of varied character and degree of seriousness occur on the tongue. In considering the differential diagnosis of these ulcers it must be remembered that early diagnosis is of paramount importance because of the relative frequency of carcinoma. It is well to consider every lesion potentially malignant until proven otherwise. Consideration of the age of the patient, history of onset and course, the presence of lesions on other parts of the body, of syphilis, tuberculosis, actinomycosis or blastomycosis, and the presence of oral sepsis and dental irritation are all of importance.

The most important lesion of the tongue is carcinoma which may occur at any age between thirty and sixty, although most frequently seen above the age of fifty. Eighty-five precent of cases occur in men. It is prone to be associated with oral sepsis and all forms of irritation.

Cancer of the tongue is most frequently preceded by a chronic superficial glossitis or leukoplakia; however, many cases occur without precancerous lesions. The site of occurrence is usually on the antero-lateral margins of the tongue. The course of the disease is one of progressive chronicity with no tendency to

heal but with rather a slow tendency to active ulceration. In appearance the ulcer has irregular margins with heaped up anemic, indurated edges sloping to an uneven base covered with dirty gray granulations. The surrounding tissues are characteristically indurated. Pain is not an early symptom, but becomes worse as ulceration gradually increases, particularly as infiltration affects the mobility of the tongue which also impairs the ability of the individual to enunciate clearly. There is a great deal of salivation and the odor becomes marked and characteristic. Metastases to the regional lymph glands occur relatively early; the glands involved are the sublingual, submaxillary and the anterior cervical chain. Biopsy confirms diagnosis.

Syphilis of the tongue may occur as a primary, secondary or tertiary lesion. The primary lesion may appear on any location in the tongue, usually on the anterior one-third of the dorsum. Negative history of exposure is not to be considered conclusive. The ulcer of the primary lesion is definite in outline; the edges are even, clean and sloping, the base is shallow and covered with a serofibrinous exudate; it is also hard and shot-like without much infiltration of the surrounding tissues. There is an associated adenopathy which is unilateral, affecting the submental and submaxillary groups. There is little or no pain, no odor, no fixation or interference with speech and very little tendency to salivation.

In secondary syphilis the ulcers may be due to the breaking down of mucous patches or to irritation of cracks or fissures in the tongue. The ulcer forming on a mucous patch is irregular and early extends through the epidermal layer. The edges are irregular but clean cut in appearance, the base being covered with a characteristic greyish membrane which can be separated. Regional adenopathy is present in greater degree than in the primary lesion. There is no pain, slight salivation, no odor and no interference with enunciation.

Tertiary lesions of the tongue are difficult to differentiate from malignancy. The ulcer begins as a gumma, with history at the onset of a localized swelling, which is painless and attracts attention only by its size; it also causes difficulty in the movement of the tongue. The gumma undergoes softening, opens and discharges, leaving a typical ulcer with irregularly thickened edges which may be undermined. The base is covered with indolent granulations without tendency to over-proliferation of epithelial tissue. There is no great tendency to infiltration or fixation. Adenopathy is present with only slight tenderness unless there is secondary infection. The tendency to salivation is more noticeable and there is also odor and some difficulty in lingual movements.

In all lesions of syphilis the serological tests are of extreme importance. In all doubtful cases particularly with regard to the differential diagnosis of tertiary syphilis and carcinoma, a therapeutic test should be made.

Tuberculosis of the mouth is a rare disease. It occurs usually as a secondary contamination in individuals who have an open pulmonary tuberculosis and is frequently associated with other tuberculous lesions of the upper respiratory

tract such as the tonsils and larynx. It has been known to occur as a primary infection. The onset comes usually in the course of a pulmonary lesion and therefore the local and general findings of a more or less advanced case of pulmonary tuberculosis are present.

Tuberculous ulcers of the tongue are prone to occur in individuals who are suffering from an active pulmonary infection or are extremely run down. The presence of oral sepsis, and particularly in those cases where there exists a primary cracking or chronic simple irritation of the tongue, predisposes to tubercular infection. In appearance the ulcers are pale and anemic. They are irregular with edges which are variously sharp-cut, beveled, everted or undermined. Usually the edges are pale, dirty and undermined, showing little tendency to bleed. The base is gray and sloughing with no induration or infiltration. The ulcers are extremely painful and there is salivation, odor and difficulty in speech and mastication. Glandular involvement, if present, eventually assumes the characteristics of tuberculous adenitis. Organisms are seldom isolated from tuberculous ulcers. Biopsy should show characteristic tubercular tissue changes.

Simple traumatic ulcers occur on the tongue—their course is usually obvious and the location associated with the point of irritation. They appear irregularly clean cut with a clean base, and without induration but a sub-acute swelling of the tongue may occur. They are extremely painful to irritation. There is no adenopathy unless they have been allowed to progress to a chronic state with the accompanying suspicion of malignant degeneration. These ulcers usually disappear after the removal of the etiologic factor. When such is not the case and the origin appears doubtful, biopsy should be performed.

Aphthous ulcers may appear on the tongue, usually in the sublingual area and in association with ulcers of the buccal mucous membrane. They occur frequently as an accompaniment of gastro-intestinal disorders and follow periods of indigestion, constipation and occasionally diarrhea. There is a bad taste, the tongue is coated and there is usually a slightly fetid breath. Generally the lesions are multiple rather than single. In appearance the ulcers are small, irregular, punctate and of grayish color. There is a fibrinous exudate and the neighboring lymph glands may be enlarged. The onset is acute and the ulcers are extremely painful, particularly to irritative substances. There is mild salivation with symptoms of gastro-intestinal disorder. They clear up quickly upon the application of silver nitrate, alkaline mouth washes and correction of the gastro-intestinal disturbance.

Ulcers of the tongue may occur in actinomycosis and in leukoplakic fissures.

An important disease of the mouth is leukoplakia, which occurs anywhere in the oral cavity, particularly on the mucous membrane of the cheek, as a thickened area with a glistening white appearance. It is related to luetic infection and is a precancerous lesion. It is seen in mouths in which the tissues are subjected to traumatism and mechanical or chemical irritation and appears to

be a condition peculiarly sensitive to tobacco. Areas which progress to the development of a fissure are to be considered already beginning malignant degenerations. Radium, X-ray and caustics are now condemned in the treatment and reliance is placed upon electro-therapy and dessication. The fissures are burnt out.

One such case occurring in the mouth of a man of middle age, where areas the size of a quarter were present on either cheek, were treated in this manner and completely eradicated.

Leukoplakia is often confused with the fungus infection—thrush. This infection is caused by Monilia Albicans and occurs frequently in adult mouths. A friend, whose mouth I had occasion to examine, told of being treated for leukoplakia for four years. The appearance of the buccal mucous membranes was of a milky, opalescent, velvety thickening. Scrapings from this membrane were examined for thrush and the monilia were found. Five per cent aqueous solution of gentian violet painted on the areas twice daily produced a cure in about two weeks.

It is interesting to note that in a recent paper, Cleveland White calls attention to the serious economic handicap to dentists such an infection may prove to be, because of the comparative ease with which the infection may be transmitted from a patient's mouth to the operator's fingers. Many of these cases are erroneously associated with novocaine dermatitis. This may be proven by culture from eczematoid eruptions on the fingers.

It is important to remember that cervical enlargements are important from a differential diagnostic standpoint. One should be familiar with the different groups or chains of lymphatic glands of the neck and the areas they drain. In any cervical enlargement it is necessary to consider the possibility of all acute and chronic conditions and also new growths. Carcinoma is probably the most common malignancy found in the oral cavity. A patient seen recently presented for the purpose of having some teeth removed because of a large submaxillary swelling which for two years had been treated as an inflammatory affair. Examination of the teeth failed to reveal the cause. In addition to the submaxillary swelling the glands on both sides of the neck in the anterior and posterior triangles were distinctly palpable and there was swelling over the parotid region on one side. A small gland removed for biopsy showed metastatic carcinoma. Further examination of the naso-pharynx revealed the source of the growth.

I recently mentioned a case, not unique by any means, in which the gums of a girl of fourteen were engorged, and swollen to the entire extent of both arches. It was thought that the patient had trench mouth, but she died ten days from onset of the first symptoms, of acute myelogenous leukemia.

A referred patient presented with a soft swelling on the inner aspect of the lower lip which had persisted for a year. There was a break in the mucous membrane and a small ulcer on the surface. It was thought to be a possible

muciparous cyst which had become traumatized but on opening, a tooth brush bristle was removed. Subsequent healing was rapid and complete.

An elderly lady presented because of a sore area on the lateral border of her tongue, which had been there for some time. Her dentist had ground and polished the lingual cusps of her molar teeth but without any benefit to the patient. She was referred because of her anxiety over the possibility of cancer. Examination revealed the fact that there were rather large triangular spaces created by the tilting of the second molar into contact with the second bicuspid because of an extracted first molar. The case was treated by placing a modeling compound splint which filled the spaces and presented a smooth surface to the tongue. The patient was comfortable in a few days. Later the second molar was removed and a bridge made to restore proper articulation.

In conclusion I wish to express my thanks to Dr. Cleveland White for his kind coöperation in presenting this subject.

# The Effect of Hypophysectomy on the Periodontal Tissues[1]

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## *Introduction and Experimental Material*

AT FIRST glance it may seem to be far-fetched to link up the effects of the removal of the pituitary gland in the rat with the problems concerned with the health and disease of the periodontal tissues. However, it may be of advantage to learn what happens to these tissues when an important endocrine gland is removed and growth is stopped and the general constitutional state is severely lowered. The periodontal tissues undergo changes and are continuous in their growth throughout life while, by contrast, the enamel stops its formation with the obliteration of the enamel forming cells and the apposition of secondary dentin slows down increasingly with age. Even if it should be proved that dentin is a storehouse of calcium and subject to calcium withdrawal, such function would still be less active in the dentin than in the calcified periodontal tissues, namely, cementum and alveolar bone.

This report is based on experimental findings in the incisors and molars of twenty-three rats in which the hypophysis cerebri or pituitary gland was successfully removed. Their age when operated upon ranged from 36 to 64 days and the interval between the operation and the death of the animal ranged from 63 to 459 days. An operation was considered successful when there was no increase in spontaneous growth for months and no spontaneous oestrous cycle for weeks or months.

A study was also made of 15 litter mate controls most of which were unsuccessfully hypophysectomized animals. The latter as well as the unoperated litter mate controls showed no indication of hypophyseal deficiency. This fact was accepted as evidence that the changes noted in the teeth were not due to operative injuries.

A histologic study of the changes revealed a number of constant deviations from the normal in the periodontal tissues. Before discussing these changes, it would be helpful to first give a brief summary of the anatomy and physiology of hypophysis cerebri and to examine briefly the changes in all the dental tissues. Just as the study of a given tooth is incomplete without studying the rest of the patient so a comprehensive understanding of the changes in the periodontal tissues is not possible without a consideration of the changes in the rest of the dental tissues. These phases are discussed in greater detail in other reports (Schour and van Dyke, 1932, a. b. c. and 1934).

[1]Read before the American Academy of Periodontology, Chicago, Illinois, August 5, 1933
From the Department of Histology, College of Dentistry, University of Illinois

The hypophysis cerebri or pituitary gland in man is an organ weighing about .65 grams. It is located in the sella turcica and consists of the anterior, intermediate and posterior lobes. Our special interest lies in the anterior lobe because its cells give rise to the growth and sex hormones. The removal of the hypophysis in the rat results chiefly in complete stoppage of growth and disturbance in the gonads. The continued administration of successive daily injections of hypophyseal extract produces gigantism and sex disturbances (Evans, 1924, 1933).

At the present moment, the hypophysis is the subject of intense research in many of the leading scientific laboratories. It is regarded by some as the master endocrine organ. It is only natural that an organ that has such a far-reaching influence on growth and that is so closely inter-related with the other endocrines should also affect the jaws and teeth.

### *Summary of General Dental Findings*

The changes in the rat incisor which is a tooth of continuous growth are: a. retardation and final cessation of eruption; b. smaller size of teeth (two-thirds normal); c. distortion of form, especially in the basal zone; d. severe histo-pathologic changes.

The changes in the rat molar which is a tooth of limited growth are: a. retarded eruption; b. smaller size of the roots; c. wide pulp chambers and apical foramina; d. histo-pathologic changes.

### *Normal Histology of the Periodontal Tissues of the Rat Molar*

Before presenting our histologic findings in the experimental animals we shall consider briefly some of the outstanding points regarding the histology of the normal rat molar. The rat molar is of special interest to the clinician because it resembles the human molar in its gross and microscopic anatomy and in its limited growth. It consists principally of dentin which is covered by enamel in the crown and by cementum in the roots. At the cemento-enamel junction, the cementum sometimes overlaps the enamel slightly by means of a fine cementum spur and often shows a hyperplasia. In the upper portion of the root the cementum is of the primary type and stains deeply with hematoxylin. It gradually thickens toward the apex where the cementum consists principally of the cellular secondary type (Figure 1, A). The cementum-apposition is continuous and so prominent at the apical end that the secondary cementum in the adult animal represents an appreciable portion of the root length (Gottlieb,

*See illustration on next page.*

FIGURE 1. Photomicrographs of mesiodistal sections of corresponding roots of upper rat molars. X 140. Al. b., alveolar bone; Cem., cementum; Den., dentin; P. D. M., periodontal membrane; Pu., pulp.

A. from rat 146 which is a litter-mate control to rat 144. Note the normal rich blood supply of the tissues.

B. from rat 144 which lived 257 days following hypophysectomy. Compare with A. Note the prominent reduction in blood supply and the deeper staining of the calcified tissues.

C. from rat 113 which lived 459 days following hypophysectomy. Compare with A. Note the numerous globules at the surface of the primary cementum; the cementicle-like structures in the periodontal membrane; the reduced blood supply and the deeper staining of the calcified tissues. See Figure 2 for magnified field.

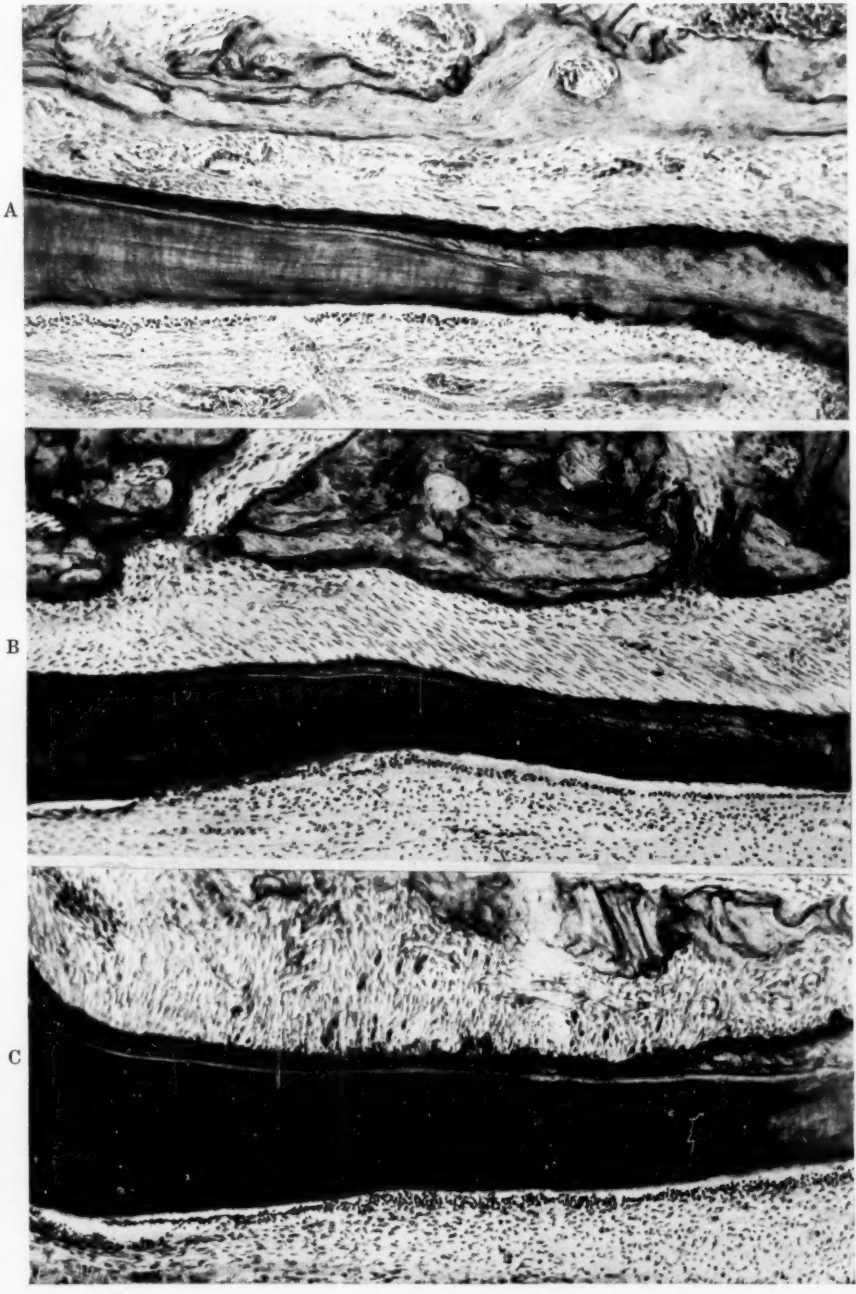


FIG. 1

1922). In addition, the apical end normally assumes the form of a knob in spite of the tapering of the dentin so that the alveolus widens to correspond with this thickening of the root end. The surface of the sceondary cementum is usually smooth. Occasionally the cementum surface at the sides of the root is sprinkled with minute granules which stain like calcified cementum.

The periodontal membrane has a very rich blood supply (Figure 1, A). At or near the cementum surface, the fibers which take the eosin stain sometimes show in cross section, small, usually round, hematoxylin staining globules or granules. These granules are seen to be confined to the cross-sectional area of the periodontal fiber. In some fibers, the granules appear to fuse so that the eosin staining portion of the fiber is diminished in area and confined to a fine peripheral border. These deep-blue staining areas can sometimes be traced lengthwise in the fibers so that they very likely assume a diminutive form of the fibers in which they are found.

We can only agree with Erdheim (1914) who suggests that these areas are possible calcified portions of the periodontal fibers. A similar phenomenon can sometimes be observed in the cross and oblique sections of the periodontal fibers as they approach and become embedded in the alveolar bone.

Epithelial rests are occasionally found in the periodontal membrane near the root surface at the bifurcation or at the apical region. They are spherical or oval in shape. Mast cells which are commonly found in the connective tissue of the rat are normally absent in the pulp and periodontal membrane.

In the control animals, the enamel that is not exposed to saliva is as a rule lined by epithelial attachment which shows no papillae. Pocket formation apparently is not common and when present is usually readily associated with some traumatic injury by a foreign body (Skillen, 1930).

One should distinguish between the histology of the normal molar of the rat and the histology of the molar of the so-called normal animal, that is the rat that is given a diet and care which at the present time is regarded as adequate. If one searches long enough in the histologic sections of the molars of the "normal" animal, one is likely to find at least in a few instances some abnormalities. The latter may be a slight resorption of cementum, particularly near the cemento-enamel junction, the beginning of pocket formation, or degenerative changes in the pulp. These abnormalities can as a rule though not always be traced to some observable causative factor, such as caries, traumatic injury of the gingivae or involvement of the periodontal membrane.

## *Histo-pathology of the Periodontal Tissues of the Hypophysectomized Rats*

### CEMENTUM

In the molar, the cementum shows resorption at the bifurcation level in the animals of shorter post-operative life. The secondary cementum shows continuous apposition and is free from resorption and yet is considerably narrower

than normal. In fact, its retarded growth is chiefly responsible for the finding of shorter roots.

In some of the animals of longest post-operative life, the primary cementum shows at the surface which faces the periodontal membrane many globules of various sizes that, like the cementum, take a deep hematoxylin stain and are arranged in the direction of the periodontal fibers. Some of these globules are arranged in a bead-like row parallel with the course of the fibres (Figure 1, C). In an oblique section of the periodontal membrane and the cementum surface of corresponding sections one can trace the globules as they appear in the center of the fibers singly or in clusters. Nearer to the cementum surface some of them appear to fuse. Immediately at the surface of the cementum, the entire cross-section of a number of the periodontal fibres appears to have taken on the hematoxylin stain. We are probably dealing here with a pathologic disturbance of a phase in the normal mode of formation of primary cementum and calcification of the periodontal membrane fibers.

There is a frequent occurrence of cementum spurs of varying lengths and thicknesses depending on the extent of the atrophy of the epithelial attachment and the length of post-operative life.

In the incisor, the cementum is thicker than normal and sometimes reaches five times the normal width. It takes a deep hematoxylin stain. In the basal zone, its surface is irregular.

The finding of a prominent increase in the thickness of cementum in the incisor might at first glance suggest a hyper-activity of the cementum forming tissue. Such a condition would be contradictory to the fact that hypophysectomy results in eventual stoppage of general growth and also to the fact that in the molar secondary cementum is formed at a slower rate than normal and does not reach a normal thickness. However, further consideration shows that there is actually no abnormal increase in the amount of cementum formed. In the incisor of the normal rat, cementum grows continuously by apposition but moves forward with the continuous eruption of the incisor. The increased thickness, therefore, in the hypophysectomized animal is merely a result of an accumulation of a tissue which would have been worn off if eruption were normal.

In the material of this investigation we find in the cementum a reaction that cannot be explained merely on the basis of retardation in growth. The resorption of cementum in the early stage is difficult to explain in view of the fact that there is evidence of apposition throughout post-operative life. The abnormal sprinkling of hematoxylin staining globules of various sizes at the cementum surface and in the adjacent periodontal membrane found in some of the animals of advanced post-operative life suggests a pathologic condition of overcalcification.

### ALVEOLAR BONE

In the animals of shorter post-operative life, the alveolar bone shows some

resorption and giant cells. In the animals of longer post-operative life, the alveolar bone presents in many fields an increased number and an irregular crowded arrangement of cementing lines. It gives the appearance of bone that has undergone an unusual amount of apposition and resorption. The bone is sclerotic and reminds one of "mosaic" bone as described by Schmorl (1932). Bone apposition is found to be continuous throughout the entire post-operative life (Figure 2).

FIG. 2

FIGURE 2. Drawing at a higher magnification of a field taken from Figure 1, Section C. Note the crowded irregular arrangement of the cementing lines of the alveolar bone (Al. b.); the numerous globules, gl., at the surface of the cementum; the osteoid border, Os; and the calcified structures, S, in the periodontal membrane.

The smaller marrow spaces of the alveolar bone tend to be fibrous and often are reduced in size. In one instance, we find in the periodontal membrane next to the alveolar crest a showering out of calcified bodies (Figure 3) that are similar to those seen more often near the cementum (Figure 2).

FIG. 3

FIGURE 3. Drawing of alveolar crest of upper molar of rat 314 which lived 110 days after hypophysectomy. Note the showering of calcified structures (S) in the periodontal membrane (P. D. M.) near the alveolar bone (Al. b.); the osteoclast (Oc); and the osteoid border (Os).

In view of the fact that skeletal growth stops almost immediately following the removal of the hypophysis, it may be surprising that alveolar bone should continue apposed throughout life. However, the apposition of alveolar bone is not at all comparable with an increase in length of the long bones. It is an expression of its high adaptability to function and stress which apparently persists even in the absence of the hypophysis. The teeth of the hypophysectomized rat except in the most advanced stages continue to perform their function although the latter is impaired. The apposition of alveolar bone particularly at the crest denotes, therefore, merely a rearrangement and an adaptation and response to the stress to which it is subjected during the movement of the tooth to function. The molars of the completely hypophysectomized rat appear to be well embedded. If the alveolar bone did not adjust itself to the tooth function by apposition and resorption, the molars would fall out because of lack of attachment.

In spite of continuous apposition, the alveolar bone in the hypophysectomized rat is smaller than normal. The less than normal amount of alveolar bone is probably a result not only of the general retardation of growth but also of the lessened stimulus following the slowing down of the growth and eruption of the teeth.

### PERIODONTAL MEMBRANE

The periodontal membrane shows a prominent reduction in blood supply (Figure 1, B, C). The epithelial rests are numerous and prominent. A number of them degenerate and become cystic and a few are calcified.

In the molars of some of the older animals, cementicle-like structures are found in the periodontal membrane that are larger and fewer than the globules found at the surface of the cementum and that react in similar manner to hematoxylin. They are spherical or oval in shape and are usually arranged in the direction of the periodontal fibers. There are also indications in the advanced stages that the periodontal fibers are at least partly calcified in the portion situated near the cementum.

In the incisor, the convex portion of the membrane, called the labial alveolar periosteum is uneven in width and often shows islands of degenerating epithelium derived from the enamel epithelium. Occasionally it also contains islands of relatively young bone, some of which is in direct contact with the calcified enamel.

In a few animals, mast cells are found in the periodontal membrane.

### EPITHELIAL ATTACHMENT

Except for some occasional slight inflammatory reaction due to trauma, the epithelial attachment is found to be normal near the gingival crest. Toward the cemento-enamel junction, however, the epithelial attachment often shows atrophy or is entirely absent. This is similar to the fate of the enamel epithelium in the anterior zone in the incisor of the hypophysectomized rat.

Where the enamel epithelium is absent, the enamel is in direct contact with

connective tissue or lined by a cementum spur or is resorbed. This resorption which was seen in seven animals may extend only into part of the enamel or may extend beyond the dento-enamel junction into the dentin. This is also seen in ground sections in which one can readily see that this is not caries.

The changes in epithelium in the periodontal tissues of the hypophysectomized rat are interesting. The epithelial attachment, which normally is persistent, tends to disappear while the epithelial rests, which are believed to be nonfunctional, increase in number and size.

### PARADONTOSIS

The association that is found in recent literature of diffuse atrophy of the alveolar bone (Gottlieb) and paradontosis (Arpa) with disturbance with the anterior lobe of the hypophysis is not apparent in the material of this investigation.

The molars are not immune to caries and show susceptibility to caries that is very similar to that of the litter mate controls. The findings of continuous resorption and apposition of alveolar bone and continuous (although slowed down) apposition of cementum indicates in spite of the presence of resorption of the root surface an adjustment which makes the firm attachment of the molars possible. It is difficult to establish the degree of resorption of the alveolar bone. Peculiarly, the resorption was less in the older animals of longer post-operative life than in those of shorter time. The height of the alveolar crest is not appreciably different from that found in the controls. The molars of the completely hypophysectomized animals appear to be well embedded. The periodontal membrane fibers are intact and their course is readily traceable.

Loosening of the teeth was not observed. The extraction of teeth for purposes of gross study was difficult. Pocket formation and downward growth of epithelium are rare and when found can usually be associated with some traumatic injury by a foreign body. In the latter cases, we are dealing with a locally produced "paradental pyorrhea" similar to but less intense than that of the rat molar described by Gottlieb (1922).

It is, of course, quite possible that the conditions in the rat molar are significantly different from those of the human molar in respect to susceptibility to paradontosis.

### DISTURBANCES IN CALCIFICATION

There is an apparent contradiction, on the basis of the histologic study of cementum and alveolar bone, between the indications of undercalcification in the animals of shorter post-operative life and the findings of overcalcification in animals of longer post-operative duration. It is, therefore, of special interest that a similar sequence of events has been found in various types of experimental hyperparathyroidism (Selye 1932, Jaffe 1933, Schour and Ham 1933) and in hypervitaminosis D (Weinmann 1933). The possibility that the disturbance of the parathyroid plays a role in the hypophysectomy picture is further suggested by the hypocalcemic condition found in hypophysectomized animals

(Evans 1933). One can readily see that considerable investigative work will be necessary before it will be possible to evaluate properly the clues found in the dental changes.

## CONCLUSION

To the clinician, the value of the dental findings in the hypophysectomy picture is two-fold: it gives us clues as to what we may expect in a hypopituitary condition in man; and it gives us clues as to what may happen in a severe constitutional set-back.

It is difficult to tell which findings are specific for hypophysectomy and which are merely a result of the severe lowering of the general constitutional state. The author, however, believes that the prominent reduction in blood supply observed in all the tissues that were studied may act as a primary factor in the symptom complex described above. The reduction in blood supply may be a specific effect of the removal of the hypophysis or the result of changes in other endocrines.

Although the findings described in this report throw considerable light on the influence of the hypophysis on the hard and soft dental tissues, one can readily see that much more research and information is needed before we can arrive at conclusions that will be of distinct clinical value. While further advance in endocrine knowledge may eventually prove to be of great value to dentistry, our present limited knowledge does not warrant endocrine therapy in dentistry.

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# The Responsibility of the Periodontist in the Social Aspect of Dentistry[1]

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THE contribution of dentistry to the welfare of the general population is usually considered as lying chiefly in the realm of the prevention of caries and this in its turn is usually visualized as being concerned chiefly with the mouths of children. When we think of the dental needs of the community or that section of it which has reached the period of adolescence or maturity we are apt to forget the preventive phase, and give more attention to the relation of established dental disease to the degenerative disorders grouped under the heading of focal infection diseases.

The Academy of Periodontology in its earlier years carried the title of Oral Prophylaxis in addition to its present title and much of its deliberations centered in the preventive phase both of caries and periodontal disease. As the science of periodontology was developed the attention of the Academy became increasingly centered on periodontia with special reference to its curative and research aspects. It is my opinion that the next step in periodontology is the study of the prevention of periodontal disease and it is, furthermore, my opinion that study in this direction is going to lead us back to the consideration of the general oral health of the child.

The above opinion has not been arrived at hastily. At the time when I accepted the directorship of the Murry and Leonie Guggenheim Dental Clinic I had become convinced that the further advances in periodontology, which were obviously needed, would have to be made through the study of those periodontal conditions in the mouths of the young which might be the forerunners of the mature periodontal disease seen in the mouths of adults. The opportunity to see the mouths of a large number of children which has been afforded through my position in the clinic has definitely confirmed this opinion. I do not wish to give the impression that I have developed a finished philosophy regarding such relationship or as regards the underlying causes of the periodontal lesions observed in the mouths of the children receiving dental care at the clinic, but I am satisfied that there is such a relationship and that we must seek a means for improving the periodontal health of the child if we are to successfully cope with the problem of an increasing incidence of periodontoclasia in the adult.

Another conviction which has been developed as a result of my observations in the last two years is that caries and periodontal disease are more closely related as regards their underlying causes than is commonly supposed. The

[1]Read at the twentieth annual meeting of the American Academy of Periodontology, August 3, 1933, Chicago, Illinois.

periodontist therefore who would interest himself in decreasing the ravages of periodontal disease must give more thought to the problem of general mouth health for the child than he has done in the past and this must include consideration of the causes of both caries and periodontoclasia. This Academy must go back to its first principles, viz. Oral Prophylaxis.

Possibly the most effective way to emphasize this is to describe some of the conditions found in the mouths of the children of to-day living in a large city as observed by a periodontist. It is hardly to be wondered at that dentists not especially interested in periodontia should give their chief attention, when studying the mouths of children, to the striking facts of existent caries. When one looks into the mouth of a child and sees numerous cavities of decay of various size and hears of toothache and the formation of abscesses it is natural to have one's attention focused on these things to the almost complete mental obliteration of the observable, but little considered gingival disturbances. This is also to be readily understood in view of the fact that advanced periodontal disease is extremely rare in the mouths of children, such lesions as are observed seeming to be quite superficial in character. The fact remains however, that gingival disease is quite as common as caries in the mouths of children. It is thus evident that when such lesions are found some defensive element is lacking and that the groundwork is already laid for a later severe and possibly intractable disease of the supporting tissues of the teeth.

It may be of interest from this standpoint as well as from the standpoint of the consideration of caries in children's mouths to give some of the figures on the latter type of disease as observed at the Guggenheim Clinic. From the time of opening the clinic to January 1st. 1933, a period of about two years, nearly nine thousand children were admitted. These children are examined in a department set aside for that purpose and are given an examination not only by exploration and inspection, but by the X-ray as well. This latter aid, universally used, has demonstrated itself to be an essential part of the examination procedure. An inspection of the throat is also made by the dental examiner who records gross deviations from the normal. An estimate is also made of the child's general condition as indicated by color, expression of the eyes, character of muscle and flesh development on the arms. This latter examination as well as that of the throat is expected to be chiefly suggestive as to the possible need for more particular medical examination.

Taking the figures for 1932 we find that of 4600 new patients examined only 117, approximately 2½ per cent, were free from caries. The ages of these children range from one to fourteen. We have become accustomed to thinking that 90 to 95 per cent of public school children are subject to caries. When we find this figure going to 97½ per cent we see the near approach of universal caries. It is obvious that as age increases, caries increases also, inasmuch as the larger number of children free from caries will lie in the younger age groups.

I have not made a study to determine at what age caries becomes universal, but this can be arrived at indirectly by following the curve established by the increase in caries in the earlier years.

At the Guggenheim Clinic an effort is made to register as many pre-school children as possible in the district served by the clinic. The following figures give a striking demonstration of the incidence of caries in the young child and also the increasing liability to caries with increasing age, the latter of course being related to the length of time the teeth are in the mouth, a seemingly important consideration:

2 years (34 patients)—
41%, free from caries
32%, 1-6 cavities
27%, 7 or more cavities
Largest number of cavities in one patient—17

3 years (151 patients)—
17%, free from caries
42%, 1-6 cavities
41%, 7 or more cavities
Largest number of cavities in one patient—20

4 years (361 patients)—
9%, free from caries
36%, 1-6 cavities
55%, 7 or more cavities
Largest number of cavities in one patient—31

Remember that while the incidence of gingivitis is not directly associated with the incidence of caries in all cases it is at least as prevalent, the difference, however, being in the degree of disease found. While in many instances the occurrence of gingival disturbance may be traced directly to the occurrence of caries, yet it may also be found in parts of the mouth where there is no caries. But it is rare to find gingival disease in a mouth in which there is no caries. This is at variance with the usual finding with regard to the relationship of these diseases in the adult mouth.

Several types of gingivitis are found in the mouths of children. Perhaps it would be more accurate to state that gingivitis is found to occur under varying conditions, the nature of the gingivitis itself showing considerable clinical similarity regardless of the conditions which seem to bring it about. I am speaking now of what I call localized gingivitis. The generalized type is a definite entity and should be considered as being quite distinct from the localized type.

Localized gingivitis is found, for instance, when proximal caries has provided an opportunity for the impaction of food against the gingivae. This occurs in the posterior regions only. An observation which I do not recall having seen in the literature and which is important in this regard is that the

spacing of the deciduous teeth which occurs as the jaw grows, providing for the accommodation of the larger permanent successors, does not usually occur between the deciduous molars, or between the deciduous second molars and first permanent molars. Here proximal contacts are maintained up to the time of the shedding of the deciduous teeth, thus affording protection for the proximal gum tissue during the heavy mastication of coarse food which is naturally requisite for proper alveolar development. The occurrence of proximal decay in this region permits food impaction, the sequential incidence of gingivitis and, because of the tenderness so produced, partially inhibits masticatory function. This is a very striking relationship and one of the greatest importance.

A very prominent cause of localized gingivitis is the gathering of food débris and bacterial colonies along the gingival line of teeth which for one reason or another are not being used for mastication. This type of accumulation is found also in mouths of adults under similar conditions, but the deposit is usually not so gross as in the mouth of a child nor is the resulting gingivitis so severe. In the child's mouth these food and bacterial accumulations rapidly envelop the entire crown of the tooth and their very extensiveness makes the viewpoint of uncleanliness as the cause of the inflammation seem to be the paramount point at issue. While we cannot minimize this factor in the etiology of the gingivitis it is nevertheless my opinion that the more important factor in such cases is that of lack of function. The two can hardly be separated, but we should keep the thought of function prominently in mind.

Another fairly common cause of gingivitis is the loosening of deciduous teeth which are about to be shed. Here we have not only the disturbance of gingival circulation due to the mobility, especially under biting stress, but also in many instances irritation of the gingival margin by the enamel edge of the tooth, where root absorption usually ceases. This sharp edge is a decided irritant to the gingival tissue. While shedding of the deciduous teeth is a physiologic process, the development of irritation during the process is fairly frequent.

It is the exception rather than the rule to find salivary calculus such as is found in the adult mouth. When it is found, however, its harmful effects are quite as pronounced as in the case of the adult. There is, however, almost no pocket formation following its deposition, the reaction being simply a gingivitis. Were it not for the fact that pocket formation in children's mouths is rare, we might be tempted to do some interesting theorizing regarding the relationship between calculus deposition and pocket formation.

Traumatic occlusion is, of course, found in the mouth of the child very frequently. Traumatic occlusion in the permanent dentition is very commonly traceable to premature extractions or of extensive caries in the deciduous dentition. These are quite as important causes as early loss of the first permanent molar. If the deciduous teeth are not lost prematurely and are kept filled, traumatic occlusion rarely develops in that dentition, or, in fact, in the permanent dentition. More and more I find myself thinking of the problem of

preventing periodontoclasia in the permanent dentition as having its groundwork in preventing the incidence of traumatic occlusion, and of the prevention of this factor, in its turn, as being founded on the preservation of the anatomical form and dimensions of the deciduous teeth. The establishment of full function with its effect on jaw development and its general hygienic influence on the oral tissues is, of course, correlative with the prevention of traumatic occlusion.

Traumatic occlusion is responsible in many cases, but not in all, for the development of gingivitis. Very commonly this, when it does occur, is associated with recession of the gingival margin and with shallow pocket formation. In some instances, however, due perhaps to superior tissue tone the gingiva, instead of developing a congestive gingivitis, assumes a hypertrophic phase, this showing itself in a decided thickening of the gum margin without any considerable redness. Traumatic occlusion, if severe, may bring about mobility of the tooth, but pocket formation is absent or is very shallow. Pocket formation of any depth is very rare in the child's mouth. When found, however, it is usually associated with severe traumatic occlusion. On the other hand, I have seen one child, a negro girl of eight years who had deep pockets on all of the deciduous molars, in which case the element of traumatic occlusion was present in but slight degree.

From the standpoint of treatment we find a very striking fact brought out, i. e., that the establishment of mouth cleanliness, the restoration of function and the stimulation of the gingivae by vigorous toothbrush massage have a remarkably beneficial effect. It is interesting to note that of these three agencies the most important is the restoration of function, the next being toothbrush massage. In fact it might almost be said that the toothbrush massage is the most important of the three. This is particularly exemplified in cases where traumatic occlusion is found and where this injurious agency, for reasons which will be discussed soon, cannot be relieved. I appreciate the fact that when stressing toothbrush massage I am also stressing cleanliness which will naturally be achieved in the course of the massage. On paper it is not easy to make a distinction, but clinical observation indicates that there is such a distinction and I regard it as important that this should be brought out.

Generalized gingivitis has already been mentioned. It occurs much less frequently in the children I have been watching than would be expected in a group whose nutritional status is probably below par as regards a large percentage of those under care. In other words, we see a large number of children whose general condition and whose economic status indicate rather pointedly that they are living on somewhat deficient diets. In spite of this fact, however, there is very little generalized gingivitis. When generalized gingivitis is found it is safe to say that the general metabolism of the body is well below par. Such cases should, of course, be given every possible aid as regards fortification of the diet and possibly other internal treatment as well. In cases showing this condition it is our experience, as would be expected, that the maintenance of

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cleanliness alone and even the restoration of function is of comparatively little benefit. Even the stimulating effects of the tooth brush are of minor assistance and such effects as are produced are brought about but slowly. Only as the general nutritional status is brought up, does the gingival condition improve.

Another periodontal disturbance which is seen with fair frequency is a generalized thickening of the pericementum, particularly as regards the permanent teeth. This seems to be not always connected with obvious traumatic occlusion. It seems more likely to be an expression of some systemic condition, the nature of which is at present a matter for conjecture. It is not always associated with visible derangement of the gingivae, hence cannot be looked upon as being the product of a marginal gingivitis. Whether it is an indication of a condition which may lead to the development of pockets in early maturity it is not possible to say, but this condition should receive careful consideration.

In review let me say that I appreciate fully the fact that I have recorded here a series of observations from which I am unprepared at present to draw definite conclusions. I have sought, however, to call to your attention some conditions which are very prevalent in the mouths of children living in a large city, with the hope that this may stimulate others to give their attention to these same conditions. About one thing however, I have come to feel very strongly and that is that dental care, including attention to the health of the gingivae should be instituted at a very early age. In the first place attention to diet and other things relating to nutrition should begin at birth, the early post-natal care being even more important than the ante-natal care. The age at which the child should begin regular visits to the dentist should be two years preferably, certainly not later than three years. This will provide for the maintenance of adequate function through the early filling of carious cavities, and the maintenance of normal height and mesiodistal dimensions of the deciduous teeth. Unquestionably much of the traumatic occlusion, to say nothing of the malocclusion without definite traumatic relationship which is observed in later life can be prevented if the deciduous teeth are restored as soon as loss of tooth substance can be detected. To wait until the child enters kindergarten is to permit, in many instances irreparable damage to the masticating machine.

I have as yet said nothing about focal infection. This is partly due to lack of exact knowledge on the subject as regards children. There are many indications, however, that the prevention of pulp disease in the child's mouth will be of tremendous benefit while, on the contrary, the establishment of focal infection very probably influences adversely many parts of the body not the least important of which are the nervous system and the endocrine chain.

I am deeply interested in the problem of diet as a factor in the dental welfare of the child, but I realize that our knowledge in that field is still incomplete. Pending the time when we have this exact knowledge and the still more remote time when we may, through educational efforts, bring about the wide-

spread application of that knowledge, I wish to assure you that local care both by the dentist and at home can accomplish a surprising amount of good and I also wish to assure you that the necessary dental care if undertaken at an early age and maintained with regularity need not be excessively expensive. It may well be undertaken, I believe, as a community health measure for all who cannot afford private dental care.

422 East 72nd Street.

## *Discussion*

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The first Academy was founded in a suburb of Athens by Plato and was both a pleasure ground and a school. Since this first Academy was founded many others have been created, for the most part, learned societies for the advancement of the arts and sciences. Not a few, however, have masqueraded under the name of academies but retained as their chief activity the essence of the pleasure ground. No one should deny that an admixture of the serious and the playful is usually desirable, but, after all, the chief function of any academy is the promotion of serious thought and in this your own association is notably successful.

The philosophy of prevention that has dominated the motives of this Academy since its inception in addition to your ambition to translate your specific interests to the general practitioner in dentistry have made this organization outstanding.

Doctor McCall has made a significant contribution to my knowledge of the subject of periodontology today. Although I am not ready to agree with everything in this excellent paper I have been impressed with the emphasis that the writer places on those factors which he has observed in children to be precursors of periodontal disease in adults. Heretofore, we have not interpreted the local gingivitis as possible forerunners of adult periodontal disease.

Doctor McCall says that gingival disease is quite as common as caries in the mouths of children. I am sure that the incidence of gingival disease in childhood does not approach the 97.5 per cent that Doctor McCall gives as the incidence of caries. This hypothesis that Doctor McCall has given; namely, that localized gingivitis of traumatic origin in the mouths of children as forerunners of adult disease, must be considered as a theory and not as a scientific fact. That is to say, that only by observing these children who have these localized gum inflammations twenty or thirty years later in their lives will we be able to state with any scientific accuracy whether these inflammations are true precursors or mere transitory phenomena.

I am anxious to agree with Doctor McCall in his conception of the scope of periodontology as stated thus: "More and more I find myself thinking of the problem of preventing periodontoclasia in the permanent dentition as having its ground work in preventing the incidence of traumatic occlusion and the prevention of this factor in its turn as being founded on the preservation of the anatomic form and dimensions in the deciduous teeth. The establishment of full function with its effect on jaw development and its general hygienic influence on the oral tissues is, of course, correlative with the prevention of traumatic occlusion." This definition of your activities means to me that periodontists are not cloistered specialists interested alone in the cure of periodontal disease by surgical or mechanical methods, but are biologists interested in the entire panorama of dental pathology as viewed throughout the entire life cycle of a person.

This definition implies further that periodontists cannot separate themselves too far from general practitioners in dentistry or from the other specialized fields.

We have heard in recent years severe criticism directed at over-specialization. Much of this criticism has been justified. Specialization is an activity in which only

mature minds of wide experience should engage. One must see the entire picture and see it clearly before he can call himself a true specialist. In the case of specialties in dentistry the specialist must understand the correlationship of his specialty with all others. He must first be a dentist before he is a specialist and before he is an effective dentist he should be a scientist trained to think in terms of general medicine and biology.

Because the problem that we have under discussion today has to do with the social responsibility of the periodontist I must add that the true specialist must be a cultured man who thinks in terms of past, present and future social and economic institutions. I am trying to say that we cannot consider the social responsibilities of the periodontist as something separate and distinct from the social responsibilities of any other intelligent citizen. In the greatest respect to Doctor McCall I am compelled to say that in this paper dealing with our common social responsibilities he has not directly stated what our problems and responsibilities are. Whether we like the idea or not we must concede that beginning on March 4 last, we have entered into one of the greatest eras of social experimentation in history. We are confronted with the fact that we have in high places a group of men who put human rights before property rights. We have been assured and we have every evidence to believe that the day of rugged individualism, the day of excessive personal profit, the day of unregulated business, is passing.

Regardless of what our prejudices are about socialization, regardless of what our preconceptions may have been, we have observed within the past few months the industrialists, the past disciples of rugged individualism, accepting an Industrial Code prescribed by a Federal Government. If there had been no abuses in the old economic order, the necessity of this government intervention would not have been imperative.

Is there an analogy between vicious industrial management and professional practice? I frankly fear that there is. We have heard in the past decade a philosophy of dental economics that was based on the business principle of extortion in charging all the traffic might bear. Fortunately, for the honor of the profession these teachings have been inspired for the most part by business men and not by dentists. These same commercial teachers have disregarded the fact that dentistry is a health necessity and must be made available to all the people. In a system of private practice it is impossible to give adequate dental care to the economic underprivileged, and there very definitely is an underprivileged class in the United States.

State medicine, health insurance, group clinics—these have been proposed to remedy this condition. Medical care for the American people must be kept under the control of the medical and dental professions of America. I wish to make it perfectly clear that unless we see the necessity for action and action now, and make voluntary effort to create an organization under the control of the profession to supply all the medical needs of the people, we may expect a socially minded government to prescribe a professional code for us. We have, then, a grave social responsibility as dentists. If we accept the responsibility we can control our own future. If we do not, we may prepare for chaos.

# Hyperemia of the Dental Pulp caused by Gingival Recession[1]

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THERE are two possible complications associated with gingival recession. The first is surface irritability of the teeth by which the exposed area is sensitive to the touch of the toothbrush, finger nail, steel instrument, etc. It may also be sensitive to sweets or acids. This is probably due to the exposure of the granular layer of Tomes, which is restricted entirely to the surface of the root immediately beneath the cementum (1). Following the recession of the gingivae a portion of the root with its covering of cementum is exposed. This latter is soon worn away exposing the most sensitive area in the entire structure of the tooth. Not all such teeth are sensitive, due probably to an abundance of buffer salts in the saliva or to better calcification, or both. In addition to surface sensitivity, there may also be reaction to temperature changes, though this latter symptom is usually dependent upon the hypersensitivity of the exposed area rather than pulp disturbance and promptly disappears following treatment. When present, it represents a merging of two distinct types of this disorder, in the first of which, surface sensitivity is the chief diagnostic symptom, whereas the second, which is the subject of this paper, is characterized by sensitiveness to temperature changes. When the latter symptom is associated with those of the first type, it is usually due primarily to irritability of the exposed area rather than to pulp hyperemia. Because this is true, they usually yield promptly to treatment needing only the proper application of some cauterant such as formaldehyde, zinc chloride or silver nitrate, to clear up both groups of symptoms.

The second type of case seen in gum recession is more serious inasmuch as the symptoms associated with it are nerve irritability and vascular changes in the dental pulp and therefore more difficult to control. The symptom complained of is always that of pain, which is more or less indefinite and intermittent. It is made worse by the introduction into the mouth of anything that is cold. A recumbent position may also increase the pain. As a rule it is difficult for the patient to locate it and in bad cases may induce facial neuralgia. An examination of the mouth will reveal some gingival recession usually involving several teeth. This may or may not be extensive, that is, the area of recession may not be large. There is usually no complaint of surface sensitivity though there may be some response to the touch of a steel instrument. As a rule it is not the symptom of which complaint is made. In the first group there are usually no definite symptoms unless the exposed surfaces are touched while in the second, pain is entirely independent of contact. A differential diagnosis can

[1]Contribution to the Committee on Scientific Research, American Academy of Periodontology at Chicago, Illinois, August 4, 1933.

usually be made by applying a pledget of cotton, saturated with ice water, to the exposed areas. In those cases in which there is pulp disturbance, the reaction will be immediate and unmistakable. This is the best way of locating the involved teeth.

In both groups, the seat of response is the dental pulp, but in the second type there is present in the pulp a condition of hyperemia, whereas, in cases of surface irritability this is not a prominent symptom. In other words there may be great sensitiveness to temperature changes without any marked surface sensitivity, and it may be absent altogether.

There are two forms of pulp hyperemia: arterial and venous. When the pulp in a tooth is disturbed as in case of thermal shock, the vasodilators bring about dilatation of the capillaries which by their expansion cause pressure on the nerves and pain follows. Immediately the vasoconstrictors react, the pressure is relieved and the pain ceases. If, however, these shocks are long continued or are severe, the walls of the capillaries are injured and are no longer able to contract under the influence of the vasoconstrictors. The result is a passive or venous hyperemia. A differential diagnosis can usually be made by the length and severity of the pain. Fortunately, there are few cases of venous hyperemia caused by gingival recession, though arterial hyperemia, often of a mild type is not uncommon. Occasionally it will express itself in the form of a chronic facial neuralgia of more or less severity. The two following cases have come under my observation within the past year and will illustrate this type of case:

## *Case Reports*

CASE 1—Mr. K., age forty-one. For over a year he had been incapacitated for work because of a nervous breakdown during which time he was under the care of a neurologist. He complained of intermittent pains about his teeth and face which were always worse at night, frequently waking him and making it necessary for him to get up. Sitting up for an hour or two, during most of which time he would smoke, afforded relief. Cold things in his mouth caused pain, but these he avoided so this was not a prominent symptom.

When he first presented for treatment he had seen several dentists in an effort to obtain relief. He reported having been treated for Vincent's infection by several and it was with this diagnosis that he was referred to me. An examination showed poor care and some gingival inflammation though this latter was not marked. Nearly all the teeth were present. There was no periodontoclasia. The gingivae were slightly receded from around most of the teeth but not to any great extent. This recession was more in the nature of an atrophic than an abrasive type. There was no history of the teeth being sensitive to the toothbrush though most of them responded when touched with an instrument. On the proximal surfaces of several above the enamel line, there was some softening of the dentine. This was in the nature of a decalcification rather than caries.

There was no evidence of Vincent's infection except the gingival inflammation and in the absence of any history of pain, this was ruled out. A diagnosis of hyperemia of the dental pulp induced by gingival recession, was made.

The mouth was protected by napkins and powdered silver nitrate was applied to the exposed root surfaces of all posterior teeth and precipitated by eugenol. They were then washed off with warm alcohol and dried, after which a thin mix of oxyphosphate of zinc was flowed over the exposed root surfaces. This required several appointments. Improvement was almost immediate; all pain ceased, his health improved and shortly after he resumed his work. Several months later after all symptoms had disappeared the softened dentine between the teeth was cut away and cement fillings made. Except for an occasional twinge of pain under strong provocation he has been entirely free from any symptoms since.

CASE II—Mr. S., age about 35, like Mr. K. had had a nervous breakdown about a year previously and was under the care of a well known nerve specialist by whom he was referred to me. He had already lost most of his molar teeth, and the gums were markedly receded on the teeth remaining though he had no periodontoclasia. Above the enamel line the dentine was softened and extremely sensitive to temperature changes. There was not, however, the marked symptoms of pain as in Case I. By the avoidance of temperature changes, and the discontinuance of the toothbrush he was relatively free from pain though his mouth was most unsanitary. Each tooth was treated as in Case I, with equally good results. His physician subsequently reported, "I don't know what it was you did for Mr. S. but the improvement in his general health is so marked as to suggest a miracle."

These two cases have much in common. Both had suffered a nervous breakdown, both were under the care of nerve specialists, both had an unusual form of decalcification of the proximal root surfaces, most marked in the second case. Both completely cleared up following treatment and both showed marked improvement in their general health.

The condition of their teeth suggests some profound systemic disturbance which may have altered the saliva in such a way as to rob it of its natural protective influence upon the teeth. This manifested itself in the decalcification of certain of the exposed root surfaces at the same time increasing their reaction to thermal changes.

CASE III—is one I saw several years ago and in some respects different from those above. The patient was a middle aged woman with a history of intermittent facial neuralgia of five years duration which was always worse during cold weather. She had consulted several physicians and dentists and had had an impacted mandibular third molar removed in the belief that it might be the cause of her pain, but without any relief. When first seen by me she had been confined to her bed for five weeks, partly as a result of the extraction, but

chiefly because of neuralgia pains. An examination revealed a generalized recession of the gums of an atrophic type from around most of her teeth. These exposed surfaces were so sensitive to temperature change that the breathing of cold air would increase the pain. They were, however, free from any surface sensitivity and there was no decay. A diagnosis of chronic hyperemia of the dental pulp was made. The exposed surfaces were cleaned with warm alcohol and dried and each was painted with a non-conducting varnish composed as follows:

Gum damar, 1 dr.
Commercial zinc oxide C. P., 1 dr.
Canada balsam, 1 m.

In the preparation of the varnish the gum damar is first liquified by dry heat and the other ingredients stirred in, after which it is poured into a small receptacle and allowed to cool. A pledget of cotton is then dipped in chloroform and rubbed on the surface of the varnish until saturated. This is applied directly to the exposed areas. The chloroform promptly evaporates leaving the teeth covered with a white glistening coat of varnish that is an almost absolute non-conductor of heat or cold.

In the case under consideration the results were little short of miraculous. Within twenty-four hours all pain had ceased, though the surfaces were kept covered by repeated applications for several weeks. She was under my care up to the time of her death fourteen years later, but except for occasional mild symptoms there was never a recurrence.

These are, of course, rare cases. I have never seen but four as extreme as these. Nevertheless they exist and unfortunately a diagnosis is usually not made and no relief given.

In less extreme forms, disturbance of the dental pulp from receded gums is more common than is generally realized, consequently the periodontist should always take this into consideration in his diagnosis and treatment of these cases.

580 Fifth Avenue.

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(1) Bodecker, C. F. "The most sensitive areas of the teeth and their operative treatment." J. Am. Dent. Asso. Vol. 14, p. 1643.

# Periodontal Symptoms Associated With Diabetes[1]

*By* Isador Hirschfeld, D.D.S.
New York City

THE fact that diabetes mellitus, like various other systemic disorders, may occasionally be indicated through the periodontium, has been called to our attention in the comparatively meagre literature on the subject. Some writers describe certain periodontal lesions in diabetics, as clinical entities that are strongly indicative of the disease, referring to them variously as "diabetic pyorrhea", "diabetic periodontoclasia", "diabetic gingivitis", "diabetic stomatitis", etc.; one writer, Williams, (1) goes a step further and divides "diabetic stomatitis" into two different types of clinical pictures; namely, "those found in patients who have well developed diabetes, but have had no diabetic treatment and those in patients who had diabetes for some time and who have been kept under diabetic control." However, from a close study of the case histories presented in the various articles, I am rather inclined to believe that in some of them, the mouth picture might be accounted for by causes other than the diabetes, which seemed to me to be merely co-incidental. Nevertheless, there are evidently some definite periodontal symptoms which are either caused by, or at least, intimately associated with, diabetes. Two such clinical entities are (1) the acute gingival abscess and (2) the sessile or pedunculated proliferations or polyps that protrude from under an abnormally heavy, usually unbroken, marginal gingiva, which they force away from the tooth surface. Either one may present in a diabetic patient and in some cases, both; however, they may also be seen in patients who apparently are not diabetics. Nevertheless, they occur so much more often in association with diabetes than without it that the presence of one, or both, of these symptoms warrants an immediate laboratory examination.

## *Acute Gingival Abscesses*

In describing the acute gingival abscess associated with diabetes, Niles (2) presents five patients in each of whom, only one tooth was involved and these were limited to the *upper* incisor group. Another writer reports similar individual abscesses, but limited to the *lower* anterior teeth. To complement these, the following cases are presented to show that gingival abscesses do not necessarily occur singly, that several may be found in one mouth; more than that, they may occur in the upper and lower jaws at the same time, in the posterior as well as in the anterior parts. Indeed, it was the presence of numerous abscesses in a mouth I observed some 8 years ago (Case I) that first led me to think in terms of diabetes, because of the frequent association of furunculosis with that disease.

[1]A Preliminary Report presented as a Contribution to the Committee on Scientific Research, American Academy of Periodontology, at Chicago, Illinois, August 4, 1933.

Case I—Miss J. W., age about 28, was referred by her dentist because of a very painful condition of the gums and teeth, which started two weeks earlier. He could not recall any previous marked periodontal manifestations. All the teeth were in position except four molars which had been replaced by pontics attached to gold shell crowns. The outstanding clinical picture consisted of a series of gingival abscesses with fistulous openings on the alveolar gingiva at the following locations: on the labial side (Figure 1), one near the lower right cuspid, another between the upper right central and lateral incisors, and a third between the upper left cuspid and first bicuspid; at various points, small subgingival polyps projected from under the free gingiva. On the lingual side (Figure 2), near the lower right central incisor there was a fistulous opening of another acute gingival abscess. The pulps of all of these teeth were vital.

All the upper teeth were loose to a degree ranging from 1 to 3 mobility, on a basis of 1, 2, 3, the right central incisor being particularly mobile, while in the lower jaw, only the incisors were loose. To minimize the pain occasioned when they were moved by the tongue or lips, the teeth had to be ligated. Except those near the upper right central incisor and the cuspid on the labial side, the subgingival pockets were not deep; the probe could not penetrate far and there was evidently no connecting space between them and the gingival abscesses. The condition developed within ten days after a severe shock due to a tragedy in the patient's family. This fact plus the sudden simultaneous development of numerous subgingival abscesses suggested the probability of diabetes, for they brought to mind the furunculosis frequently associated with diabetes. *(The patient was entirely unaware that she had diabetes.)* A urinalysis at this time disclosed the presence of 5.3% sugar. The blood sugar record is not available. Nothing was done locally aside from ligating the teeth, and the patient was placed on insulin. Within several weeks, the gingival swellings were greatly reduced, all the fistuli closed up, except the one over the upper right central incisor; the teeth became considerably tighter; and the acute pericementitis disappeared completely. A subgingival scaling was done and it was found that there was comparatively little subgingival calculus. Further periodontal improvement was noticeable after the patient carried out proper hygiene in which she was thoroughly instructed (Figure 3). Unfortunately, the patient discontinued the use of insulin for ten months during which time she did not come to the office. When she returned (Figure 4), new gingival swellings were in evidence, also pain. The lower central incisors had been extracted and the other teeth were much looser than at the first visit. The gums had become a bluish red and three gingival abscesses had formed over the upper left incisors and cuspid and another opposite the lower left cuspid and first bicuspid. The palatal sides of the upper anterior teeth had deep pyorrheal pockets.

I had occasion to see this patient again three years later at a metabolism clinic, where she was under treatment for diabetes. At this time, all the teeth had been extracted.

FIG. 1

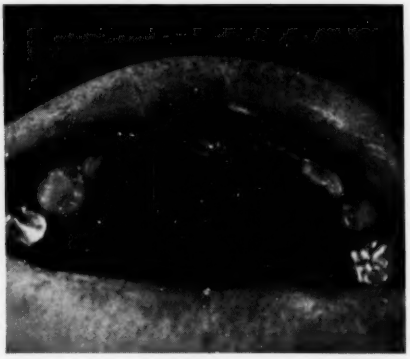

FIG. 2

FIG. 3

FIG. 4

FIG. 1—Case I—Photographed at the time the patient was first seen, showing gingival abscesses with their fistuli near the lower right cuspid, the upper left cuspid and between the upper right central and lateral incissors; also the subgingival polyps protruding from underneath the gum near the upper right lateral, and left central, incisors, between the upper left cuspid and bicuspid, also, distally to the lower right cuspid. The pulps of all teeth were vital. Note also the ligature placed over the extremely loose lower anterior teeth, to minimize pericemental pain produced by pressure upon them with the lip or tongue.

FIG. 2—same as Fig. 1—lingual view, showing gingival swelling and fistulous opening near the lower right central incisor.

FIG. 3—same as Fig. 1, six months later—no gingival abscesses nor subgingival proliferations are present although no dental attention had been given. The improvement was evidently attributable directly to the treatment of the diabetic condition, and later on, to the partial return to function and hygiene, now made possible through the disappearance of the pain.

FIG. 4—same as Fig. 1, photographed 10 months after the discontinuance of insulin and other anti-diabetic measures. The lower central incisors had been extracted. New acute gingival abscesses had developed, the fistuli of some of which are readily discernible over the upper left incisors and cuspid. Similar gingival abscesses presented between the lower left cuspid and incisor, and the first and second bicuspids.

Case II—Mrs. I. L., age 48. The main complaint was a very painful condition of the gingiva and a recent pericementitis about the lower anterior teeth. For three years, the patient's urine had been kept sugar-free through diet. However, during the year following, the diet had not been watched and at the time

she presented with the acute gingival condition, the sugar was 3%. There was a large gingival abscess (Figure 5) with a fistulous opening labially to the right lower lateral incisor and cuspid and one between the lower left central and lateral incisors. The former had become progressively larger during a period of about ten weeks while the latter had occurred twice previously, disappearing each time without any special treatment. Another small abscess presented lingually to the lower right central incisor, midway on the root. As in Case I, the pulps of these teeth were vital. Submaxillary glands on both sides became tender after the appearance of the swelling. The hypertrophied papilla between the upper right central and lateral incisor, too, was quite tender. In general, the gingivae were purplish in some parts and cyanotic in others; they were quite thickened on the palatal side. Underneath them, a generalized, highly advanced pyorrheal condition presented, also on the labial and lingual aspects of the lower anterior teeth. The lower teeth and the upper central incisors and right cuspid were particularly loose. For an entire year, the lower central incisors were too loose to function.

She was referred back to the physician for treatment. Soon thereafter the patient received several prophylactic treatments, including a partial removal of the subgingival deposits. When I saw her again, three months later, there were no gingival abscesses although the teeth were still loose and showed evidence of gingivitis and pyorrhea. Eighteen months later (Figure 6), I found that all the lower teeth had been extracted and that a hard swelling of the gingiva presented labially to the upper right cuspid over a deep pyorrheal pocket. At this

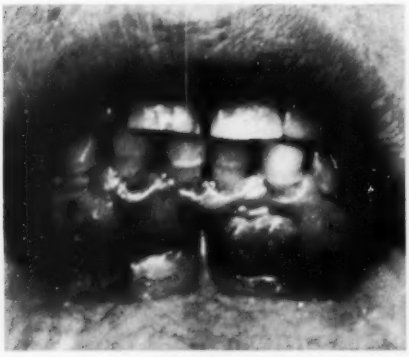

FIG. 5

FIG. 6

FIG. 5—Case II—note two acute gingival abscesses. The one on the right side shows one fistula, indicated by the dark spot, and another below it, by the white speck of pus which exuded at the time of photography. The abscess opposite the left incisor is quite sessile and was "pointing" at three places. The ligature on the lower teeth was placed to minimize their excessive mobility and so reduce the extreme pain caused by the acute pericementitis when the teeth were moved by the lips or tongue. The pulps in all the teeth were vital.

FIG. 6—same as Fig. 5, 18 months later, showing the swelling of the gum opposite the upper right cuspid as a result of an acute gingival abscess recently developed. The pulp in this tooth was vital.

time, the patient had not had diabetic treatment for a year, and urinalysis showed 1½% sugar.

*Subgingival Proliferations*

The subgingival proliferations or polyps, like the acute gingival abscesses above referred to, are found in diabetics only occasionally, according to my private clinical experience and to observations made by me in a survey of a group of patients in a metabolism clinic; nevertheless, their apparently greater incidence in association with diabetes than without it seems to justify the suspicion of the presence of the latter disease even as does a furunculosis, which, too, may occur without the systemic condition but usually calls for a test for sugar.

The proliferations are, as a rule, either salmon, brick, old-rose, and in some instances, bright red, in color. Protruding from under the gingival margin, they tend to push the gum away from the neck of the tooth, thus producing the appearance of a marked gingival hypertrophy. There is, however, a tendency for the marginal gingiva itself to be abnormally heavy and to remain unbroken by the expansive force of the polypoid growths. When the gum is not quite so markedly displaced, the discoloration of the protruding proliferation gives the impression, at first glance, of a marginal gingivitis but upon closer observation, it is soon evident that the deeply discolored tissue is an additional growth and that the original gingiva is practically unaffected by the abnormal color.

Clinically, the proliferations simulate those of pregnancy perhaps more than any other clinical entity but they may be differentiated as follows: in the latter, the polypoid tissue is more likely to be pedunculated rather than sessile; is less likely to force the gum away from the tooth surface; *is more often localized interproximally* and *the gingival margin is more apt to be frayed,* producing a sort of dog-eared effect; besides, there is the associated history of pregnancy.

Case III—Dr. L. H., age 42, dentist, came to my office with a painful condition of the lower anterior gum, presenting (Figure 7) two semi-hard swellings near the left and the right lateral incisors; also an acute pericementitis about the lower incisors. At these points, the heavy marginal gingivae were noticeably forced away from the teeth by subgingival brick-red, inflammatory tissue. A similar tendency was evident about the other teeth, particularly the upper left central incisor and on the palatal side of the upper first molar and lingually to the lower right second bicuspid. In general, the gingivae were of a bluish tinge. The height of contour of the gingival margins on the upper left palatal side was milky white—often seen in heavy smokers, but this patient did not smoke. There was a generalized, advanced pyorrhea in various parts of the mouth.

Some time prior to his visit to my office, the patient had sought treatment for the pyorrheal condition which he had then first noticed. After several

treatments, the gums began to swell, and there was a tendency to gingival abscess formation with a constantly increasing acute pericementitis of the lower incisors. He had not been able to eat with these for a month before this visit.

The prolonged acute pericementitis, the gingival abscess formation, and the brick-colored subgingival proliferations readily suggested diabetes *of which, however, the patient was entirely unaware.* The suggestion of a probable diabetic condition caused the patient to remark that during the past six months he was

FIG. 7

FIG. 8

FIG. 9

FIG. 10

FIG. 7—Case III. Note the swollen gingiva in the lower incisor region. (This was actually much heavier than is indicated in this photograph taken at the time of the examination.) The marginal gingiva is forced away from the neck of the tooth by the subgingival proliferative tissue. A similar tendency is evident on either side of the upper left central incisor.

FIG. 8—same as Fig. 7, one week later, showing marked improvement of the lower gingivae as a result of treatment for diabetes alone—local treatment being purposely withheld.

FIG. 9—same as Fig 7, showing still further improvement in the lower gingivae, still without any local treatment. The upper gums, too, are considerably improved but they had received some treatment.

FIG. 10—same as Fig. 7, showing generally healthy appearance of the gingivae two months after the institution of routine conservative periodontal treatment, including an improved toothbrushing technique.

continually thirsty, urinated frequently, tired easily, and had lost about 12 pounds—(altogether he had lost 18 lbs. during the past year)—all symptoms of diabetes. A blood examination and a urinalysis made, accordingly, the next day, showed 7.59% sugar in the urine and 250 milligrams of sugar per 100 cc. of blood.

The patient immediately placed himself on a special diet and a regime of sufficient exercise. Within a week, the blood sugar was reduced to normal and the urine showed no sugar. The systemic symptoms, including dryness of the mouth and fatigue, were no longer in evidence. No local treatment of any kind was given so that I could follow the changes in the periodontium upon the systemic improvement alone. The patient coöperated scrupulously with me in this respect. At the second visit, one week later (Figure 8), the pericementitis had entirely disappeared, suppuration had ceased, and the gingival swelling and discoloration had become less marked. (Before the institution of the systemic treatment, these had all been becoming progressively worse.) A month later (Figure 9) the swelling opposite the central incisor was almost all gone and that over the lateral incisor, was markedly reduced. The general color of the hypertrophied gingiva formerly cyanotic was now almost normal. No change in the method of brushing had been made. At this time, the patient was taught an improved toothbrushing technique and routine conservative periodontal treatment was undertaken with the result that resolution took place very rapidly (Figure 10).

Case IV—Mr. L. P., age 28, electrician, had been under more or less continuous treatment for diabetes for about three years, and when seen by me was receiving insulin three times daily. The blood sugar was 288 milligrams, and the urine showed ½%. For several weeks prior to his visit to the dental clinic, the gums bled upon brushing for 2 to 3 days at a time. The condition was diagnosed by his dentist as a subacute Vincent's infection for which he was then treated several times and a sodium perborate wash prescribed. There was marked improvement as far as the clinical picture of Vincent's infection was concerned. However, at the time I saw the patient (Figure 11), salmon-colored subgingival proliferations protruding from under the gingival margin, thus forcing it away from the necks of the teeth, were evident in many parts of the mouth. These were not tender, and had, no doubt, developed prior to the onset of the Vincent's infection. The gums were generally swollen and somewhat cyanotic, especially in the lower anterior part of the mouth (Figure 12). Heavy subgingival deposits were found under the proliferations.

The patient explained that the urine was usually clear of sugar, that he adhered strictly to the diet continuously for about four weeks and then relapsed for a time, and that the condition of the blood sugar and urine fluctuated accordingly. He is also tubercular, X-rays of the chest showing cavitations. However, the subgingival proliferations above described cannot be attributed to the tuberculosis nor to the Vincent's infection as the clinical symptoms of

both are of an entirely different character and localization. Only the right side was used for mastication, and it seemed entirely unaffected. These symptoms disappeared with routine conservative treatment of chronic gingivitis.

FIG. 11

FIG. 12

FIG. 11—Case IV, showing heavy, lower gingivae partly pushed away from all the incisors and the right cuspid by salmon-colored proliferations, not unlike those in Dr. L. H. The slight discoloration of the marginal gingiva over the upper right lateral incisor is due to the presence of a small polyp under the gum (located by a probe).

FIG. 12—same as Fig. 11, showing salmon-colored subgingival polyps protruding from under the unbroken marginal gingiva which is thus partly pushed away from the teeth. Note the edematous, cyanotic appearance of the gum in this area. Most of the teeth are fairly tight. A generalized pyorrheal condition, labially and lingually, is in evidence, with pockets of varying depths, and a considerable amount of heavy serumal calculus. The gingival condition improved readily upon local treatment.

Case V—Mrs. I. S., age 30, mother of 3 children, had diabetic treatment for two years, but this was discontinued during the year preceding her visit to the dental clinic for treatment of a gingivitis, which was especially marked on the left side, where the upper first and second molars and all the lower teeth, except the first bicuspid, were missing. The marginal gingivae were inflamed, bluish, somewhat heavy but unbroken, and were forced away, in most instances, from the tooth surface by partly protruding subgingival proliferations, brick, to bright red in color. This was especially marked on the upper right central incisor, left central and lateral incisors and first bicuspid, labially (Figure 13), and on the palatal side of the upper left central incisor and second bicuspid. Similar conditions presented on the lingual side of the lower left central and lateral incisors.

In addition, the patient had a generalized incipient pyorrheal condition and there was a fistula about half way down the root of the left central incisor. The large masses of protruding tissue on the labial side of the upper left first bicuspid and the upper left lateral incisor, bled very easily upon compression.

The patient did not return for treatment until 15 months later. The teeth were now considerably looser and subgingival proliferations were evident in many new locations (Figures 14, 15 and 16). It was especially interesting to

note that there were marked subgingival polyps on the palatal sides of the upper left second bicuspid and second molar which had had no opponents for over 3 years; also that although there was a marked traumatic occlusion on the upper left first bicuspid, no proliferation had developed there—a fact which indicates that traumatic occlusion is evidently not an important factor in the production of these proliferations. During this interval, the sugar in the urine ran up to

FIG. 13

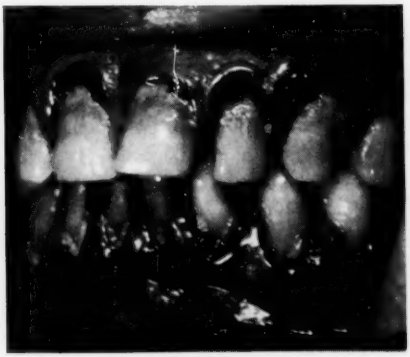

FIG. 14

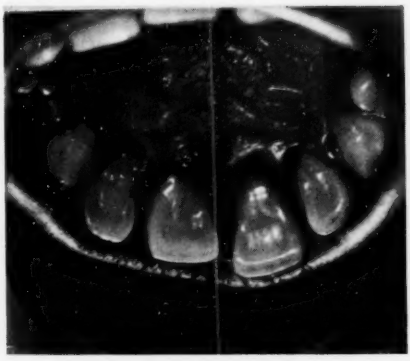

FIG. 15

FIG. 16

FIG. 13—Case V. Case showing subgingival proliferations especially marked over the upper left lateral incisor and the first bicuspid, and smaller ones over the upper central incisors.

FIG. 14, same as Fig. 13—15 months later. No local treatment was instituted; in the meantime the diabetic condition, too, was neglected. By comparison with the previous illustration, it will be noted that the teeth which were most affected in the latter are now less so, and vice versa.

FIG. 15—same as Fig 13, palatal view, showing proliferations from under the unbroken marginal gingivae which they forced away from nearly all the teeth.

FIG. 16—same as Fig. 13, showing proliferation on the palatal side of the second bicuspid and the second molar. These teeth had no occlusal opponents when the patient first presented, and according to my diagrammatic record charts there were no proliferations; the fact that they have developed since then without the restoration of the missing lower teeth points definitely to the fact that traumatic occlusion is not an essential element in the localization of this lesion.

as high as 7%. The patient claims that the teeth became looser as the percentage in sugar increased and conversely, that they tightened somewhat when there was a general systemic improvement. When she was seen again half a year later, there was a large swelling extending across the median line on the palatal side of the upper right third molar and a similar one, but smaller, on the lingual side of the lower left lateral incisor. Both were painful and no pus could be drained from them through the pyorrheal pockets.

The above histories of patients who show gingival symptoms associated with and very probably caused by diabetes have been selected for presentation from a larger group observed and recorded by the author. The following, however, is a case which particularly merits mention, for it is an outstanding instance of the importance of *early recognition of these gingival symptoms by the dentist:*—

Case VI—Miss S. W., age 23, a clerk, presented at a labor union clinic for the examination and treatment of her mouth. Dr. Esther Graubart, the periodontist in charge, who was also associated with me in the Periodontia Clinic at the Hospital for Joint Diseases, saw the subgingival proliferations above described and accordingly suspected a diabetic condition. She advised the patient that a urinalysis should be made before any periodontal treatment could be instituted. The patient, highly sensitive, was indignant at this suggestion, as *she looked and felt generally healthy,* and expressed a reluctance to visit a physician. She was again urged to have the urinalysis made, and once more she returned to the clinic without the required report. Dr. Graubart then refused to treat the patient, who subsequently complained to the director of the clinic. After investigation, he persuaded the patient to follow the periodontist's advice, and soon thereafter the report was received showing 4% sugar. Her blood sugar, too, was found to be abnormally high, (245 mgms. per 100cc.) and she was hospitalized for insulin therapy and observation with relation to a special diabetic diet in keeping with her means.

According to the hospital case history chart, her physical examination at the time of admission showed "a well-developed and nourished girl, with no pertinent physical findings". During the three months immediately prior to the discovery of her systemic condition, the only subjective symptom was constant thirst. It was found that her mother, too, is a diabetic. The patient, now grateful for the care she received, relates that while she was in the hospital, the physician in charge repeatedly pointed her out to visiting practitioners as a patient whose diabetic condition was first noted through her dentist's recognition of the gingival symptoms.

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# The Significance of a Complete Laboratory Report in the Classification and Treatment of Some Periodontal Cases[1]

*By* JAMES A. SINCLAIR, D. D. S.
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WHEN it is realized that many people harbor numerous bacteria and that only a few actually develop infection, we are brought face to face with the fact that there is something peculiar about some people that permits them to escape while others succumb to the infection.

This "something" is that protection in the body which is called "immunity." It might also be expressed as a lack of susceptibility to bacterial invasion or the power of resistance which enables one to avoid disease.

The human race possesses a natural immunity which renders the invasion and growth of microörganism difficult. This immunity, however, is not absolute. It may be broken down or reduced by exposure to cold, hunger, fatigue, trauma, or shock.

In our cures we are successful in stimulating the body to fight the disease with its own weapons. In our failures we do not raise the body resistance sufficiently. To cope with this situation we should have an understanding of the conditions lowering the resistance. Many obscure conditions may be revealed by urinary analysis, metabolic tests and blood examinations, including blood chemistry.

The examination of urine yields information of great value, and should be made a part of the examination of all patients who do not readily respond to treatment.

The diseases of metabolism most frequently observed are gout, diabetes, acidosis and alkalosis. The characteristic feature of such diseases is disturbance of regulation in the process of maintenance and growth, and this pathogenesis is based, in part, on disturbance of the glands of internal secretion whereby possibly the nature of catalizers is altered or the supply of normal catalizers is increased or decreased to an abnormal degree, and in part on deficiencies or excess of any of the food factors.

Diabetes is characterized by a diminished capacity to utilize glucose, by an excessive accumulation of glucose in the blood (normal 58-110 M. G. M. per 100 c. c.), and by the presence of glucose in the urine.

In cases of periodontal lesions in untreated diabetes, it is almost impossible to obtain satisfactory results: Satisfactory results may often be obtained by the use of insulin.

[1]Contribution to the Committee on Scientific Research, American Academy of Periodontology at Chicago, Illinois, August 4, 1933.

*Significance of the Results of Blood Tests for Acidosis*

Two tests have been applied to the blood for the detection of acidosis:

1. Determination of the carbonate content of the blood—the so-called alkaline reserve.

2. Determination of the hydrogen-ion (pH) content of the blood. The latter merits little consideration because the results obtained by its use show little or no deviation from the normal except in moribund cases. Indeed, it appears very likely from animal experimentation and from theoretical considerations that any sustained elevation in the actual pH of the blood is incompatible with life.

Since the bicarbonate content of the body fluids represents the most important alkaline reserve of the tissues, it is apparent that this first line of defense will show deviations from the normal, in conditions of acidosis. It is well established that the blood bicarbonate is depleted in acidotic conditions caused by fixed acids. As examples, we may refer to the production of unoxidized fatty acids, like B-oxybutyric and aceto-acetic in diabetes, and the improper elimination of acids produced in normal metabolism as happens in some cases of nephritis.

In our practice we carry out a routine procedure that enables us to solve many obscure conditions that we are satisfied are the cause of slow or nonhealing lesions.

In all cases of periodontal lesions we obtain the percentage of hemoglobin. If it is below 70, a red blood count is made. This will indicate the severity of an anemia and in some cases the class of anemia.

In all complicated cases in addition to obtaining the percentage of hemoglobin and the red and white cell count, a urinary and blood chemistry examination is made. If a leucocytosis of 10,000 or a leucopenia of 5,000 cells or less is found, a differential count is made.

*Leucocytosis (Musser Internal Medicine)*

The coccal infections uniformly produce leucocytosis. This fact is interesting not only as representing the reaction of the circulating blood, but as also representing the local reaction in the tissues which the body makes to the coccal infections, and, accordingly, this type of infection is usually known as a pyogenic infection. In a very definite way the leucocytic response is connected with the capacity of the individual to recover from an infectious disease. A failure to produce leucocytosis is regarded as a bad omen and the production of vigorous leucocytosis is always looked upon with satisfaction by the physician. Changes in the type of granulocytes are also regarded as important. Increase in the number of "stab" leucocytes makes the same unfavorable impression as it does in other infections. The leucocytosis terminates with the infection and, therefore, seems directly connected with the mechanism of recovery. Recovery depends on many factors, none of which can be established as the

most important. In a general way, antibody formation as expressed by agglutinins and precipitins of the blood is looked upon as one of the important agencies in the production of recovery. Such antibodies may also be bactericidal or antitoxic.

### *Phagocytosis*

At least two of the main groups of white blood cells possess phagocytic powers; there is some doubt as to the lymphocytes. The neutrophil leucocyte is the active agent of the phagocytosis of bacteria, while the monocyte exhibits amazing phagocytic powers in the engulfing of colloid and particulate matters. It is quite possible that these are among the chief functions of these cells.

In pus and exudates it is easy to find evidence of the phagocytic powers of these cells for bacteria, but only rarely does one find bacteria within the leucocytes of the circulating blood. McCutcheon (Am. Jour. Phys. 1923, 66, 180 and 185) has measured the rate of ameboid movement of the different varieties of leucocytes. The granular leucocyte is the most active and travels across the field at a rate of about 30 microns per minute; the lymphocyte moves much more slowly. The rate of movement is influenced by many factors including temperature; this suggests a greater activity of leucocytes during acute infections, and a less activity during a chronic infection.

Dr. Chas. Mayo says (collected papers of the Mayo Clinic, XXIV, No. 32, p. 628), in speaking of infected tonsils, "The small infected tonsil after having been infected earlier and having developed immunity temporarily is no longer enlarged, is not even red, has a few large vessels, but are the more dangerous type, for they contain small surface pus pockets and are without reaction to pain. In fact, the capacity of the blood to react is exhausted, the number of leucocytes is reduced from 7,500 to 6,000 or 4,000, although the presence of infection calls for an increase in each cubic millimeter of blood. To put it in another way, the policemen of the community have become used to living with criminals and bootleggers and no longer react."

If there is no change in the blood picture of our chronic periodontal diseases then we have an abnormal blood picture and should regard it as such. The normal becomes abnormal. With the presence of infection we should have a leucocytosis instead of the normal number of white cells.

As a result of the excellent work of Schilling, we now have a new interpretation of the part played by the white cells in infection. With the Schilling differential count it is possible to obtain greater diagnostic aid from the blood and also to frequently anticipate changes in physical signs.

According to Schilling, there are three main groups of white cells, namely, granulocytes, which are increased in acute infections; monocytes, increased in sub-acute infections; and lymphocytes, which are increased in chronic infections. The role of the granulocytes in disease is the main factor in interpreting a differential blood count by Schilling's classification.

The polymorphonuclear neutrophilic leuocytes, commonly called "polys," are divided by Schilling into four groups, namely, mature, or segmented forms; stab, or almost mature cells; juvenile, or immature forms; and myelocyte, or the youngest form of all neutrophiles. As the blood picture shows a relative increase in the young forms, the change is spoken of as a "shift to the left" or a shift to the immature form of "polys." The other granulocytes, namely basophiles and eosinophiles, along with lymphocytes, and monocytes (mononuclear or transitional cells), are the same in the Schilling as in the Ehrlich differential count.

By arranging the list of cells and count in a horizontal line from left to right, it is often possible to see at a glance the condition of the patient. Such arrangement is called a hemogram. From left to right the order is basophiles, eosinophiles, myelocytes, juveniles, stabs, segments, lymphocytes, and monocytes. If an imaginary line is drawn between segmented and stab cells, an increase in cells to the left indicates an increase in infection. When stabs alone are increased, Schilling refers to the change as a degenerative shift to the left, while an increase in juveniles and myelocytes, representing more impression on the bone marrow, is spoken of as a regenerative shift to the left.

Changes in cells other than granulocytes also have much significance. An increase in basophiles indicates loss of resistance. The eosinophile is the most sensitive of all leucocytes, and is first to disappear in acute infections. Both lymphocytes and monocytes have a tendency to decrease in acute infections, and to return when the infection is overcome. A sudden rise in monocytes is spoken of as the dawn of convalescence.

After the monocytes are increased, if the patient still improves, a change to the more mature "poly" takes place. This is known as a shift back to normal or to the right. A high shift to the left with a high monocyte percentage indicates a good phase of defense with the struggle (infection) still raging. Following a shift to the right, a favorable sign is reappearance of eosinophiles and an increase in lymphocytes. This last finding indicates a recovery.

Another index of severity of infection is the nuclear index proposed by Gerard and Boerner. By dividing the segmented or mature forms of "poly" by the three young forms the nuclear index is found. They consider a nuclear index of 15 or more as normal for adults, an index of 10 representing a slight infection, of 5 a very severe disease, and below 5 an extremely severe infection.

It is difficult to estimate accurately the incidence of the various types of blood diseases which are encountered in the practice of medicine, but undoubtedly, chronic secondary anemia is the condition most frequently observed. This is true because so many different conditions are responsible for this variety of anemia. Chronic blood loss due to uterine disorders, hemorrhoids, bleeding peptic ulcer, neoplasm, improper diet and almost any chronic long-continued disease may cause a secondary anemia. Various other anemias, such as pernicious

anemia which have been considered somewhat rare in the past, have appeared to be more common within recent years. This is probably because the condition is now recognized with greater accuracy, due to improved diagnostic methods and a more widespread knowledge concerning the disease.

Apparently various blood diseases fluctuate greatly in frequency as the result of unknown causes. One of the mysteries of medicine is why a disease such as chlorosis which was so common in the past, has now almost completely disappeared. Within the past few years at least two blood diseases appear to be increasing in frequency—idiopathic microcytic anemia, which occurs for no obvious cause in women of middle age, is now a relatively common disease, and agranulocytic angina has only recently been recognized as a clinical entity.

### *Anemia Secondary to Infection*

All but the most trifling infections tend to lower the red blood cell count and hemoglobin percentage. Often the anemia is mild and transitory, but certain infections characteristically bring about severe anemia and even those infections which, as a rule, do not, will do so if they are of long duration or of unusual severity.

All infections of long duration will bring about anemia, but certain infections, including acute rheumatic fever, the septicemias, streptococcus viridans endorcarditis and other streptococcic diseases, syphilis (perhaps in part from treatment), and the spirochetal diseases as a group, exhibit a special tendency toward severe anemia.

The excellent work of Pepper and Farley (in "Practical Hemotological Diagnosis, 1933) gives the most modern view of the blood conditions in the various diseases. Some of these conditions are interesting from our point of view.

### *Tumors*

Anemia—By no means do all patients with neoplastic disease exhibit anemia; not even all those with carcinoma. Only when additional factors come into play does the blood count become abnormal.

### *Pregnancy*

Often in mid-pregnancy a mild anemia of the secondary type develops and sometimes this becomes severe, but as a rule the picture always remains that of a secondary type of anemia with low color index, moderate morphological changes in the red blood cells, and a moderate increase in the formation of these as estimated by an increase in the young reticulated red cells.

### *Malaria*

The anemia of malaria is primarily brought about by the destruction of red cells by the plasmodium. Even after a short acute attack some anemia may be present and in more chronic cases the anemia may become intense. In these severely anemic cases there may be superadded an element of diminished blood formation.

Intestinal parasites especially the hookworm (uncinaria) and the fish tapeworm (Dipyllobothrium latum) may cause marked anemia. In infestation with the former the picture is always that of a secondary anemia no matter how low the red cell count and hemoglobin percentage may fall. The color index is always low. As a rule the anemia is not intense, but occasional cases exhibit red cell counts below 1,000,000 and hemoglobin below 20 per cent; eosinophillia is a constant finding.

*Syphilis*

Very early in the secondary state of syphilis there appears a moderate anemia of secondary type; this is believed by Stokes to occur more often in women than in men. The hemoglobin may fall as much as 20 per cent. This anemia differs in no respect from the usual secondary anemia incident to any infectious process. If it tends to progress to a more severe stage, it suggests that the syphilis is following a definitely severe course, or that a complicating nephritis has developed.

An increase of white cells up to 12,000 and even to 20,000 is not unusual in secondary syphilis; this rise is always due to an actual lymphocytosis; the lymphocytes forming 50 per cent. or even over 60 per cent. of the increased total count. Stokes states: "The combination of moderate secondary anemia with lymphocytosis in women always calls for a Wasserman test and a careful search for clinical signs."

Congenital syphilis exhibits marked anemia only when there is distinct visceral involvement. Perlman and Wright conclude that the secondary anemia present in congenital syphilis has no peculiar features separating it from secondary anemias associated with chronic infectious disease of any types. Frank reports in such cases a white cell count of 30,000 due to an enormous increase in lymphocytes up to 90 per cent. of the total. No true relationship between syphilis and actual leukemia has been demonstrated.

*Tuberculosis*

There are so many complications associated with all stages of tuberculosis that the blood examination is practically useless except the differential count of the white cells and the sedimentation rate of the red cells. Usually the total count is normal. Except in advanced stages associated with secondary infection, a count of 12 to 16,000 may be observed.

*Idiopathic Anemia With Achlorhydria and Microcytosis*

This syndrome closely resembles chlorosis and also the anemia from chronic blood loss.

Clinically this condition is found in adult women before the menopause. A lack of hydrochloric acid and the presence of a stomatitis may at first suggest that the anemia is of the Addisonian type. However, the blood picture is distinctly not that of the Addisonian anemia. Perhaps it should be considered

simply an anemia secondary to achlorhydria for this seems to be its most constant feature.

Blood Picture—The red blood cell count is only moderately reduced usually to between 3,500,000 and 4,000,000. The hemoglobin, however, is found as low as 30 per cent. and occasionally even lower. As this would lead one to anticipate, the color index and volume index are very low and the red cells themselves are far below the normal size. There is little variability in shape; reticulocytes are not increased; nucleated red cells are rare.

White cells are apt to be below normal in number due to a reduction of neutrophilic leucocytes, and the platelets may also be somewhat low. The prompt benefit from adequate administration of iron is so constant as to be accepted as one of the diagnostic features of the disease.

### *Agranulocytic Angina*

Agranulocytic angina is a disease of unknown etiology, characterized by chills, fever, marked prostration, angina, a remarkable reduction in the polymorphonuclear neutrophil cells of the peripheral blood and usually a rapidly fatal course.

There is an associated ulcerative lesion involving the mucous membranes. This usually occurs about the mouth or throat, but may involve the anus or vagina.

The striking feature is a pronounced leucopenia which may progress to such an extent that the polymorphonuclear neutrophil cells almost completely disappear from the peripheral blood. The reduction at first is only in the granulocytes, but later the lymphocytes and monocytes are also diminished. There is no alteration in the red blood cells unless the illness is prolonged, and then an anemia with a low color index may develop. The platelets are usually normal or increased except when the illness is prolonged and they may then become diminished.

The use of hydrochloric acid in a variety of infections, especially those characterized by pus formation, has been recommended by Granville Hanes (1), Burr Furgurson (2), Norman Smith (3), W. M. Salter and C. D. W. Colby (4), as a means of stimulating leucocytosis and promoting phagocytosis.

I have been fortunate in being familiar with the work of Dr. Colby in his attempts to stimulate cellular activity in pulmonary tuberculosis which was complicated by suppurative processes. In cases of asthma, hay fever, acne, sinusitis and common colds, the results have been encouraging.

We have used this method in a number of cases with promising results.

To emphasize the value of stimulating leucocytosis in a case that would not respond to the ordinary treatment, the patient, a school teacher, upon examination presented the following picture:

Urine examination, negative; Wasserman, negative; diet satisfactory; no complications that we could discover.

10 cc. of hydrochloric acid, 1 to 1,000 dilution, was given intra-venously once each week for a period of eight weeks. The white cell count was taken before administration of the acid and one twenty minutes after. The hemograms (see following page) will show the increase of leucocytes after each injection. The results in this case are very encouraging and we hope permanent.

The blood picture is that of a chronic infection,—a leucopenia and not a leucocytosis.

The so-called pyorrhea pocket, before treatment, is without the body tissue, that is, it opens into the oral cavity. It is subject to the fluids, food particles and débris of the mouth. Food for the bacteria is present. When operated upon and the diseased tissue removed, though some bacteria remain, the pocket becomes within the body tissue. It is sealed with coagulated blood and a wall of defense completely surrounds the infected area and through the phagocytic action of the white cells the remaining bacteria are destroyed, as well as any necrosed tissue that may be present. This is what happens in the *successfully* treated pyorrhea lesion.

The humoral defense mechanism against bacteria or their products usually found in the mouth is set up from the earliest or first invasion of the microörganisms. The reaction or antibodies are produced as the bacteria slowly increase. If this were not true any of the many injuries to the gums, cheeks, tongue or palate would result in an acute infection. The bleeding gums would be an entrance for the bacteria and an acute infection would follow. It would be dangerous to use a toothpick or a stiff brush. Thus we see the humoral defense in the form of antibodies present in the blood serum, together with the cellular defense of the leucocytes, both operating to control the poisonous effects of bacteria.

Once the patient has acquired a condition of pyorrhea any thing that lowers the resistance, has a marked effect upon the cure, arrest or re-occurrence of the once active lesion.

Pyorrhea is not a disease of youth, though youth is not exempt. As age advances the occurrence of the disease increases, so also does the re-occurrence of once arrested cases increase, until finally as old age arrives there are but few who are not edentulous as the result of the final breaking down of the supporting tissue of the teeth.

During the period from youth to old age the same symbiotic group of microörganisms are always present. In youth they appear to be present in greater number, due to neglect or uncleanliness, by which there is a lowering of resistance. It seems, therefore, that a change has taken place in the patient, rather than in the bacteria which may be present.

Many cases of lowered resistance, severe anemia, diabetes, nephritis, tuberculosis, and syphilis are found to free of periodontal lesions.

The continued presence of the same symbiotic group of microörganisms or their products in constant contact with same tissue, will sooner or later

**Hemogram Schilling** **Date Nov. 26th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| 71 | 11.4 | .7 | 4.649.000 | 5400 | 0 | 2 | 0 | 0 | 3 | 51 | 40 | 4 |

**Hemogram Schilling** **20 minutes later** **Date Nov. 26th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 6.350 | 0 | 1 | 0 | 0 | 4 | 65 | 26 | 4 |

**Hemogram Schilling** **Date Dec. 3rd, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 9.500 | 0 | 1 | 0 | 0 | 2 | 53 | 36 | 8 |

**Hemogram Schilling** **20 minutes later** **Date Dec. 3rd, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.600 | 0 | 1 | 0 | 0 | 1 | 62 | 25 | 11 |

**Hemogram Schilling** **Date Dec. 10th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 8.000 | 0 | 1 | 0 | 0 | 0 | 66 | 30 | 3 |

**Hemogram Schilling** **20 minutes later** **Date Dec. 10th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.850 | 0 | 1 | 0 | 0 | 1 | 70 | 20 | 8 |

**Hemogram Schilling** **Date Dec. 17th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.850 | 0 | 1 | 0 | 0 | 0 | 69 | 25 | 5 |

**Hemogram Schilling** **20 minutes later** **Date Dec. 17th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 11.800 | 0 | 1 | 0 | 0 | 0 | 74 | 20 | 5 |

**Hemogram Schilling** **Date Dec. 24th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.250 | 0 | 1 | 0 | 0 | 1 | 60 | 28 | 10 |

**Hemogram Schilling** **20 minutes later** **Date Dec. 24th, 1932**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 11.000 | 0 | 1 | 0 | 0 | 1 | 67 | 24 | 7 |

**Hemogram Schilling** **Date Jan. 1st, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.200 | 0 | 1 | 0 | 0 | 0 | 60 | 35 | 4 |

**Hemogram Schilling** **20 minutes later** **Date Jan. 1st, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.950 | 0 | 1 | 0 | 0 | 2 | 64 | 30 | 3 |

**Hemogram Schilling** **Date Jan. 7th, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.000 | 0 | 1 | 0 | 0 | 1 | 56 | 37 | 5 |

**Hemogram Schilling** **20 minutes later** **Date Jan. 7th, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.750 | 0 | 1 | 0 | 0 | 0 | 71 | 20 | 8 |

**Hemogram Schilling** **Date Jan. 14th, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leukocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 9.800 | 0 | 1 | 0 | 0 | 2 | 58 | 30 | 9 |

**Hemogram Schilling** **20 minutes later** **Date Jan. 14th, 1933**

| Hemoglobin | Hemoglobin Gms. Per 100 cc Blood | Color Index | Erythrocytes | Leucocytes | Basophiles | Eosinophiles | Myelocytes | Juveniles | "Stabs" | Segment | Lymphocytes | Monocytes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Neutrophiles | | | | | |
| | | | | 10.900 | 0 | 1 | 0 | 0 | 1 | 73 | 20 | 5 |

produce in that tissue a sensitization to the bacteria in the lesion resulting in a breaking down of the tissue so changed. The condition continues as long as the bacteria remain and the forces of immunity continue to be depressed. The result of the tissue change is always the same in every individual. The condition is allergic in its tendency and the rapidity of the development and extent of the injury depends upon the degree of the sensitization of the tissue to the bacteria present.

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# A Philosophical Approach to Periodontia

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It has now been several years since I have appeared before this Academy, to take part in its discussions and to listen to those who have addressed its audiences. It was with a feeling of sincere pleasure that I accepted the invitation of the chairman of the Program Committee to make this address. During the years of my absence from the meetings, my interest in the subject of our mutual concern has not abated, nor has my interest in this Academy lessened. I think, however, that my absence during the past few years has given me an opportunity to contemplate the activities within our science, which we call Periodontology, and to reflect upon its status and development, in a way which would not have been possible had I always been present. I shall endeavor therefore to put the essentials of periodontia's position in their proper historical and sociological perspective and to outline clearly the reason why periodontia exists as it is understood today.

The world views this Academy as a scientific body—its members meeting as an aggregation of scientists intent upon the advancement of Periodontology. I have prepared this address with the object of examining this field of interest, in order to present for your consideration my personal views.

The term "periodontology" was coined by the founders of this organization to provide a title or caption for our chosen field of study, under which the mental product of the members could be known to the scientific world; it is the name of the science under which a periodontist practices his art. Among the first activities of the associated members, was the problem of providing a name to replace the very vague, unsatisfactory pathologic terms, "pyorrhea" and "pyorrhea specialist", which were then in common usage. The term "periodontoclasia" was first adopted as a term to supplant "pyorrhea alveolaris". Following this, was adopted the term "periodontist", to designate and to supplant the name "pyorrhea specialist". The term "periodontology" evolved naturally. Its suffix "ology" carries the thought of discourse, and, as in all young organizations, talk and discussion are inevitable, it appeared essential that there exist a word to designate this branch of dental science.

In order to make it clear to the minds of the younger members of the Academy, who have adopted the cognomen "periodontist", as a designation of their special field of service, it would be well to review briefly the hereditary descent of the specialty of periodontia, from its earliest days to the present.

If St. Appolonia is recognized as the patron of dentistry, due recognition should also be given to Riggs, for it was he who laid the cornerstone of our knowledge of

periodontology. Yet even before the days of Riggs, a dentist was a specialist—but all of dentistry was his specialty. But with the discovery, by Riggs, that the disease, later known as pyorrhea alveolaris, would yield to sufficiently skillful ministrations, there developed, one after another, a small group of dentists who called themselves "pyorrhea specialists". The designation "pyorrhea specialist" endured to the year when this Academy was founded; the large majority of pyorrhea specialists of that day adopting the newer name, periodontist. The change, however, was one of name only. Actually, the art of the pyorrhea specialist was continued under the name, periodontist.

Riggs' discovery was, that when accretions found upon the roots of teeth suffering from pyorrhea, were adequately removed, the operation was followed by a definite healing reaction within the affected area. This reaction was observed to be a biologic trend in the direction of health; specifically a cessation of the flow of pus. It should be stated at this point that pus, flowing from the border of the gingiva was then regarded, not only as the outstanding but as the one single characteristic symptom of the disease. It was small wonder then, that the operator believed that the objective of his work was the suppression of this pathologic symptom. While we now recognize other symptoms and other pathologic phenomena, as being of equal importance in the symptomatology of periodontal disease, it may truthfully be said that the same mental attitude, regarding case management, is still resident in the minds of most periodontists of today, viz., that their objective is the suppression of disease symptoms.

All treatment of disease is predicated upon the assumption that a pathologic process exists; for without the presence of observable disease, there would be no rational object in applying treatment of disease. The art of treatment does not necessarily imply that healing and health will follow; for treatment is validly so called, even though the biologic reactions which it is hoped will result, do not actually eventuate. Treatment must, however, have an objective. And it is the objective of periodontic treatment which I wish to discuss with you.

An objective point can be said to be the ultimate object of an exertion. Acceptable treatment usually implies exertion. Whether or not it connotes physical action, it should at least imply mental clarity. Whenever the mind is set upon a definite objective or destination point, the intention of the individual is usually lucid and intelligent. What I wish to call to your attention is the fact that the present day periodontist has an objective, but he has inherited it from the pyorrhea specialist. And unfortunately, he has inherited the identical conception of the pyorrhea specialist, whose objective in treatment was to attain the suppression of the pus flow from the region of the alveolar process. The mind of the pyorrhea specialist was clear upon this point, for he held only this one matter as important. What shall we say today regarding the intelligence of the periodontist who holds the same objective, although his knowledge of the nature and progression of periodontal disease has been so vastly extended? Are we to go on being satisfied with a conception of periodontic treatment based upon the objective of a suppression of active symptoms of disease only? And shall we be satisfied with such a result when this objective is attained? If not, what then is our ideal?

It is clear that an ideal exists only in the imagination. An objective, then, is entirely conceptual. What then is the highest conception of the ideal toward which we periodontists may work? *The true objective of every member of the "healing art" is Health.* When we limit our objective to the suppression of the symptoms of disease only, our aim is too low.

Associated with the word health are such adjectives as hale, healthful, hearty, hygienic, sanitary, salutary, sound, strong, vigorous, well, and physically wholesome. On the other hand, associated with the word disease are such adjectives as affected, ailing, complaining, disordered, indisposed, ill, infirm, sick, unhealthy, and physically unsound. These are the antonyms of health. There is a decided gap between these and the words designating health. We cannot conscientiously aim at the lower ideal.

For the past twenty years the composite mind of this Academy, and in fact the whole of dentistry, has centered its interest on the subject of disease. The outcome of

this concentrated mental effort in dentistry has been the science of dental pathology. Pathology is that branch of biologic science that treats of morbid conditions, their causes, nature, etc. No man can deny that pathology is science or claim that it constitutes unessential knowledge. But if one were able to learn all that is yet known about disease, one's knowledge about *health* would nevertheless remain the same as it was in the beginning. For pathology teaches us nothing about HEALTH.

It is not logical to state that the pursuit of knowledge of disease will provide a working basis for the development of bodily health. To hold to such a sophism would be equal to stating that regular attendance at symphony concerts were all that was needed to develop great musicians; or that learning to read would necessarily teach one how to write. If you desire to know disease, by all means study pathology, the science devoted to it, but if you can admit that the attainment of health for your professional clientele is your objective in practice, it would be well to know first the science devoted to that objective. Health today has its own science, toward which, unfortunately dentistry has not as yet been attracted. There is a biologic science which relates to the maintenance of health. Its name is Hygiene—not dental hygiene, but *Hygiene*. If health is the objective of the healing art, it would be well if the members of this Academy should acquire knowledge of the science of Hygiene and make the pursuit of health its objective.

There is at the present time a growing popular demand for health. Especially is this true regarding the health of the teeth and mouth. I am sure that the interest of this Academy would quickly center upon health and the means for attaining it, if its members would realize that this is their real objective, and that the study of disease is only desirable insofar as it may be made useful in helping to devise ways of attaining *health*.

Lest I be misunderstood, I may say at this point, that while the objectives of periodontia have been undergoing a change in my own mind, the intrinsic operative acts used to attain these ends have changed but little. Riggs depended chiefly on the scaler in his treatment of pyorrhea, or scurvy of the teeth as he called it, and the scaler we still have with us and always will. The toothbrush has come down to us from a period still more remote, and it still remains a necessary part of our armamentarium. Grinding or filing of teeth for the relief of excessive occlusal stress, has been advocated at various times for more than one hundred years. These items of clinical procedure are still employed in my practice. The important point is this—the mental picture of what I am trying to achieve by using these same therapeutic agencies, has changed. And it is my desire to express to you, that the same instrument or the same technique in principle, may do good or harm according to the way used; or better still, according to the intelligence, which means the mental concept and the objective of the individual, which guides it.

Actual health has rarely been discussed from this forum in the past fifteen years—not, in fact, since the Academy changed its name. It is doubtless an almost forgotten fact that the original name of this organization was, The American Academy of Periodontology *and Oral Prophylaxis*. Whatever the reason may have been for the deletion of the last three words, it would be interesting to speculate on the psychological change which accompanied the modification. The word Prophylaxis properly belongs in the domain of pathology, for we are told that a prophylactic is an "operation intended to ward off something—*especially disease*". Certainly no one ever attempted to ward off health. If a prophylactic were understood to have *health* as its purpose of application, it would be so defined in dictionaries. Prophylaxis is a word having a negative implication, yet it carries more of the thought of health in the phrase, oral prophylaxis, than is to be found in the term periodontology, with its present frankly pathologic significance. I cannot but feel that to drop this part of the name of the Academy reveals a mental step away from, rather than toward the Academy's true objective.

It is true that when this change in name was made, the science of dental pathology was just beginning to be developed. The members of the Academy and dentists in general, looked to this new development with the belief that this budding science would soon solve all their problems. Much knowledge and culture has come from the

new dental pathology. But there has been disappointment also, for the development of health through treatment of disease has not advanced hand in hand with the advancing knowledge of disease.

It is approximately only twenty years since actual dental pathology, as a science, was created. I refer to the work of Black who also presented to dentistry its first dental Anatomy. This gave to dentistry its two first basic sciences, *which are all that dentistry possesses today.* There are still to be developed at least two more dental sciences, essential for the further advancement of dentistry into a valid branch of the healing art; these are Dental Hygiene and Dental Physiology. There is still a paucity of scientific information regarding the *functions* of the teeth and other oral tissues. No scientific treatise comparable to Black's Special Dental Pathology has as yet made its appearance under the title of Dental Physiology.

So far as "*dental*" Hygiene is concerned, the more moderated the criticism of this, as the name of a science, the less likely the disgrace of dentistry will become a public scandal. The fact is apparently not widespread, judging from dental literature, that what dentists today dignify as *dental* hygiene, is actually *pseudo*-hygiene. The premises under which dental hygiene is written and published, and its art practiced, gives to it no right to the already preëmpted name Hygiene—"the branch of medical science which relates to the preservation of health". It should be enough to state that in the entire science of Hygiene, as expounded by those who in any degree comprehend the subject, there is no mention of a cleansing process. Yet, if the topic of the cleansing of the teeth were deleted from the literature of present day *dental* Hygiene, there would be nothing left but the punctuation marks to show that the subject once had words to express a mistaken thought. All body cleaning processes are to be found under the caption Sanitation. Hygiene and Sanitation are not synonyms, any more than are arithmetic and geometry.

The statement that hygiene does not possess a cleaning process is *strictly true;* it may be misleading, however, to those dentists who understand today that cleanliness of the mouth or cleaning teeth *is dental* hygiene. This should not be interpreted to mean that the teeth need not be cleaned, or that a plan for developing health of the mouth may omit efforts at cleaning. The reverse will always be true, for the reason that when health wanes in the mouth, filth accumulates upon the teeth. Oral disease is always filthy, and filth must be removed by all who are practically interested in *oral health,* if the health of the teeth is to be preserved or promoted.

I have already stated that the objective of all medical and dental effort, within the healing art, is Health. This holds, of course, for the Periodontist. How then can the periodontist accept credit for achievements which have not resulted in health of the entire mouth? And without health of the entire mouth, the end result of periodontic case management may properly be called a failure. It is not becoming of me to point the finger of scorn at other men. In fact, it is not the failures of other men I have in mind. Of these I know but little, and I speak of them less. But I cannot forget my own failures and of these I have a perfect right to speak.

If you, my fellow members of this Academy, can agree that the incentive which impels you to continue at your work of periodontia, is oral health, the same as it is mine, there is a great future for the American Academy of Periodontology. For there must be mental growth and development of periodontia's basic principles, to the point of their ideal perfection; and there must be case management-achievement, or this Academy will eventually become a social club, comparable to "The Mystic Knights of the Sea". There *must* be growth, there must be *life;* and all life is change. It is high time the Academy passed into its own Renaissance and into a new period of its mental development.

We periodontists have long centered our interest in disease, but our interest in disease should be contemplative and cultural. I would not *know* less about disease, nor relax my interest in pathology, but I would know more about the *science of health,* and the biologic mechanism within the body which maintains the equilibrium of health, for oral health is my objective.

Periodontists have from the beginning of their history recognized the need for know-

ledge. Periodontia, however, is an art. The source of knowledge of an art, such as the treatment of periodontal disease as it is today believed to be, must lie in *some* science. Call it by the name of Periodontology if you will, but if the objective of a periodontist is oral health, it is obvious that the science devoted to an understanding of *health*, must be the source of knowledge which can liberate and perfect the art, which is devoted to the promotion of *health*.

As a specialty of a profession, designated as "a branch of the healing art," we periodontists do not know very much about the science, Hygiene, aside from its name. And the knowledge which we *think* we possess, is largely a mass of misinformation. On the other hand, we, as dentists, *knew* very little about the science of pathology, either dental or medical, aside from its name, in the year 1900. With the development of the science of pathology directed at studies relating to the teeth, we came to believe that dental pathology was actually our working science. Had our objective in periodontia been the development of *disease*, instead of the development of *health*, dental pathology might have been successful as our working science, and who among us would dare deny it. But health has at all times been our objective, whether we comprehend the fact or not, even during the years devoted to the treatment of disease. And it is our objective today.

When a biologic art, such as periodontia is seen to be, is in dire need of the possession of *four* sciences as a minimal requisite, and there exist but *two* sciences upon which we can build knowledge for our "branch of the healing art", there is small wonder with this paucity of science, that there is mental confusion, regarding our daily tasks. There would exist the same mental confusion in the minds of our children in the elementary school grades, when seeking knowledge pertaining to learning letters, to reading, writing, or to the subject of arithmetic, if any two of these fundamental subjects of preliminary education were omitted in the school. Would it not soon become apparent that the education of our children was deficient? That is the situation in which periodontia is seen to be today.

When the mind is centered upon the study of disease, it can be said that the individual is "majoring" in Pathology. In such an event the subject of Etiology and Diagnosis of disease receives high attention. Dental Pathology has succeeded in differentiating disease according to its clinical and histo-pathologic characteristics. Regarding the several clinical types, we more or less agree upon a name for each and to this consideration, we give the pathognomonic designation, "differential diagnosis",—a very euphoneous term. We tabulate upon our diagnostic charts the details of the pathologic conditions as found in the mouth of a patient, priding ourselves upon our ability as dental diagnosticians. But after pathologic diagnosis, how do we employ our findings; what use do we make of them?

No man having mental pretentions, even if these pretentions are not of the highest quality, can enjoy making himself mentally ridiculous before the world. A desire for human respect is innate in us all. But of greater value to one's character, however, is the respect in which a real man holds himself. To discover that one is being mentally ridiculous before *himself*, even though the world may not have discovered the fact, brings a sense of shame to the ego and the human conscience. It was a condition similar to this in which I found myself regarding my daily office work.

Like most of us, my interest had become centered in disease. I saw myself, so I thought, fast becoming a dental diagnostician. Greater attention and detail was being paid to my diagnostic charts and to bacteriological smears than to the health welfare of my patients. One day I propounded these questions to myself:—"What practical use for the patient are you making of all these clinical records?", "What benefit accrues to the patient from all this rigmarole?", "Is it true that your advancing knowledge of pathology is hastening the health welfare of your patients?". All these questions disturbed my peace of mind, for I was obliged to acknowledge to myself that the thing which I had been visualizing, as pertinent to the "treatment of disease" could not rationally be called treatment of disease. My conscious interest had been centered in pathology, and therefore in disease, but my subconscious hope and case management had ever been *in health*.

With a realization of these facts, came

the necessity of making a survey of my own operating procedure. The question arose, "After these years devoted to the study of dental pathology, in what way and to what extent have your acts of treatment been advanced, due to the knowledge of disease which you have gained from the study of pathology?". After an elaborate notation of symptoms of disease upon my charts, after the compilation and listing of the several kinds of bacteria recognized as being present in the patient's mouth, the practical knowledge of how to suppress all these had advanced in no particular degree because of the possession of the charts. And the interesting revelation became apparent that the so-called plan of treatment remained *consistently the same* in all cases, regardless of the pathognomonic designation of types which the charts revealed. In other words, it was discovered that the name given to the disease-type made no change in the manner of approach to its management. Again the question arose, "If the acts of treatment are actually the same for all oral disease types, why go to all this bother to differentiate between the several types?". A fair question!

With these perplexing questions came a fundamental question, "What is your objective in your work," "What is the name of the science under which you proceed?." Periodontology of course, but what does the name mean? "Dental pathology is a study of dental disease and this has influenced your advancement but little. You say health in behalf of your patients is your objective when you treat disease. What then do you *know* regarding the science of health? What is the science of health, what is its name, and how can you make it apply to your daily work?" The answer came promptly. *Hygiene* is the name of the science devoted to the understanding of health, and self examination revealed that I not only knew little on the subject, but after investigation what little I did know was found to be untrue. This conclusion was reached after a review of the present literature of dental hygiene was placed in comparison with the text books on the subject of hygiene, as that science is recorded by accredited scientists. But my astounding discovery was that hygiene deals only with the biologic reactions *within the body* of the patient. I had thought it dealt with *cleanliness,* like most of you.

For many years I followed diagnostic procedure, and in my office files there is still to be found a tremendous bulk of diagnostic charts toward which today my mind gives not the slightest interest. The tabulating of clinical distinctions between one pathologic picture and another, upon a chart, as found in a single individual mouth, is both an interesting and a cultural diversion. I am convinced, however, that these records have slight, if any, bearing upon *health. And I issue a friendly challenge to anyone, to explain in print* the pertinence which differential diagnosis has upon the oral health of the individual patient, who is about to be treated for some oral disease. It is *the subject of health* which should hold our professional interest, not that of *disease.* It is the science of Hygiene which devotes its interest to Health. Pathology has always devoted its interest to understanding disease. And rightly so. Disease is pathology's subject.

There are many dentists who know little of hygiene, yet who readily believe that hygiene is not a dynamic pursuit, but rather a weak science devoted mainly to keeping the health of those who are already healthy. Nothing can be further from the facts. The knowledge which the science of hygiene gives, can of course be directed toward the development of greater degrees of health, as well as toward the development of health for those who are already healthy. Hygiene makes every phase of bodily health its central theme, and its fundamental concepts apply equally to the entire body, as they do to the health of the teeth.

There is a biologic mechanism within the body which functions solely to the end that the life of the organism may be preserved. This innate faculty operates within the mouth, as well as elsewhere in the organism. The art of hygiene devotes its entire interest to the problem of motivating this natural impulse. When disease enters into the clinical picture, this life-sustaining mechanism is found to be functionally maladjusted. So long as life exists in the organism, the health mechanism has some degree of efficiency, however slight; but the more advanced the state of disease, the more is its operation interfered with. The recovery

and repair of disease *within* the body is termed healing; a biologic process; a body function. But with the teeth carrying the burden of defective function, the pathologic state cannot unaided, be overcome. The biologic mechanism, or function of health, may be said to operate inversely, and in direct proportion, to the degree of the magnitude of disease.

It has already been stated that the essential difference between the art of hygiene and clinical efforts directed at the suppression of the symptoms of a pathologic lesion, is simply a matter of mental perspective and approach. The approach through hygiene centers upon the effort to release the mechanism of healing, so that it may function and proceed with its biologic duties of repair. The true hygienist, seeing physical clinical pictures of disease, proceeds in his art with a fore-knowledge that if the injury to the tissues is to be repaired, the change must occur through the action of the body process—*Healing*. His interest, therefore, is centered in the living cells adjacent to the lesion, not in the dead and decadent cells within the lesion itself. Hygiene is concerned with the adaptive reactions which occur in the body, viz., "The potentialities of living protoplasm," innate, for purposes of retaining life. And its progressive sequence is, *from* disease, *through* healing, *to* health.

In the science of pathology, the approach requires that the dentist be learned in the knowledge of the nature of disease, and that he speculate upon its causes. These speculations, pathologists dignify with the term *Etiology*. The interest in this approach is supplied by the phenomena which the clinical picture of disease exhibits. "What causes it?" is asked. The horizon of the perspective is confined to the limitations of the site of the pathologic cells. The mind of the true pathologist does not stray outside of the limits of the lesion. He finds no interest in healthy tissue, for an understanding of disease and its manifestations comprises the entire scope of his activities. This statement is not made in disparagement of pathology or of pathologists. Each science has its own field of study, and pathology is devoted wholly to a study of morbid physical suffering. Interest in pathology begins only after health has receded, and disease has gained ascendency.

Hygiene develops its theme in one direction only, viz., towards health,—and regardless of the physical state of the body at the moment. Pathologic studies proceed in the reverse direction, viz., *from* health, to disease and through the whole course of disease, to death. And even after death, the pathologist holds interest in his subject. The post-mortem is always interesting. Not so the hygienist. Disease does not arouse interest only as it becomes a barrier to health.

The fact should be apparent that dentistry, possessing no written science of dental physiology and no valid science which can be called dental hygiene, has been sustained upon a scientific diet of pathology alone, and that it is at present suffering from a dietary deficiency for want of adequate science aliment. An examination of contemporary dental literature would reveal that constant mental-food injestion of this single phase of dental science has resulted in dentists actually *specializing in pathology*. And we know that they occasionally succeed in this.

A synonym of the word science is knowledge, and knowledge is a clear and certain apprehension of truth. The essence of living is to keep alive on earth, and this being accepted as true, it is essential that the body of man shall possess health. General health and dental health are identical, for health is a bodily condition, the same as is disease. Both are dependent upon the life of the organism and both cease when death occurs. When disease occurs, however, the health mechanism, which is the sole interest of the science of hygiene, breaks down. To possess health, not only of teeth but of the entire organism, it is essential that we periodontists know something about the nature of health and its biological laws. Periodontists are still intent upon the subject of disease and its progress, due to their futile attempts to overpower disease. Such misdirected efforts can find their complements only in the impossible picture of preliterate man, fixing attention upon the cold climatic conditions surrounding him, rather than upon his own body's needs for heat and protection. Were health today

pursued with the same intensity of action as that which is directed at disease, we might well be able to point with happiness and with pride upon our accomplishments of developing a healthy race of men possessing a full complement of healthy teeth.

The wise man realizes that the difference between good and evil is, in essence, the same as between health and disease. Such a thought can be stated only relatively, however, yet positive active forces are sponsored by life, and they alone are continually supported by an external and Eternal Will. Health determines life, and disease terminates life, as does death. I implore you to set your minds upon a science which can provide physical life worth living—Hygiene, the science of Health.

# A Method of Procedure In Equalizing Distributions of Stresses.

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IT IS a recognized fact in dentistry that traumatic occlusion is an important factor in the development of dental and periodontal disease. The investigation of this subject by Stillman, McCall, Box and others has brought about an important change in the progress of preventive and curative dentistry. It has aided not only in the treatment of periodontal disease, but its influence has become an important factor in the progress of all phases of dental science.

The purpose of this article is to simplify the procedure in obtaining equal distribution of stresses in natural dentures. Before this can be properly planned a definition of the terms to be used is most advisable. Traumatic occlusion (1) may be considered as a functional incoördination of the teeth and relates wholly to a mechanical disharmony. It produces stress in excess of what a given tooth is physically able to withstand. The pathological reaction which follows, that is, trauma, is not traumatic occlusion. This is termed occlusal trauma. It is a biologic reaction to stress, a reaction to injury, and is clinically expressed as a pathologic effect. Traumatic occlusion relates wholly to the occlusion, while occlusal trauma relates only to the injury. Traumatic occlusion (1a) may be observed either in centric occlusion, that is, the relation of the inclined planes when the jaws are closed in the position of rest; or, in eccentric occlusion—the relation of the inclined planes of the teeth when the jaws are closed in any of the excursive movements of the mandible. The closing of jaws, whether in the act of mastication or in the act of bringing the upper and lower teeth into contact, results in the application of a varying degree of occlusal force to the teeth. The extent of normal occlusal force varies with each individual tooth according to its function, and each tooth in the normal jaw is provided with alveolar support sufficient to withstand the stress that is normal for that tooth. Normal occlusal force may be defined as the innocuous distribution of the stress or stresses exerted upon the teeth in their various contact relationships.

Recently, Box (2) has urged the change of the term traumatic occlusion to traumagenic occlusion. (3) The term has since been changed to traumatogenic occlusion. I shall quote from his article:

"In the fields of surgery, medicine and pathology, under the term traumatic, the lesions and conditions cited, according to the accepted usage of the word, are always the result of trauma. This meaning and usage of the word traumatic, therefore, is at complete variance with that commonly understood in the term *traumatic occlusion*. According to the accepted and proper usage, the occlusion is always the result of trauma, while in the present dental nomenclature the trauma is the result of the occlusion. In other words, the meanings are diametrically opposed.

"Cases are to be seen in every practice where the occlusions exhibited are directly the result of trauma. Various examples were noted in the author's practice during the past year. Fracture of the mandible, with resulting disturbed occlusion, axial displacement of the upper central incisors through violent impact, and loss of the crown-tips through injury, resulting in a changed occlusion, are examples of cases manifesting definite *traumatic occlusions*. Many other examples could be cited. It therefore is obvious that there is a place for the term *traumatic occlusion* in dental nomenclature.

"The problem of trauma, i. e., injury produced mechanically in occlusions of natural dentures, under biting pressure, takes into consideration a new factor in the study of occlusion viz: the relationship between the tooth and its supporting structures. In normal physiological occlusion, the relation between the tooth and its periodontal tissues is such that, under occlusal stress, no injury is produced in them. And, further, the tissues are best able to resist harmful agents of various kinds which bring about pathological changes in the periodontium.

"It can hardly be denied from the evidence of the present day, that in certain occlusions, under biting pressure, traumatic changes occur in the supporting tissues of the teeth. To these occlusions, the writer has given the term *traumatogenic*.

"There are presented, then, in clinical practice, occlusions which are clearly the result of trauma, and occlusions which produce trauma. In applying to the first class the term *traumatic*, and to the second, the term *traumatogenic*, in which the meanings conveyed are completely opposite, it would appear that terms have been chosen which designate these conditions more accurately, thus making for greater clarity in dental nomenclature.

"A traumatic occlusion, therefore, is a contact relation of the masticatory surfaces of the teeth which is directly the result of trauma. A traumatogenic occlusion, as applied to natural dentures, is an occlusion which, under biting pressure, produces an injury in the periodontal tissues."

The term *traumatogenic* will, therefore, be used in the remaining portions of this paper.

Balanced occlusion as interpreted by prosthodontists cannot be applied to the natural teeth. This can best be exemplified when protrusive relation is referred to. In artificial dentures, at least three points are in contact in protrusive position (three point contact), and in this position a balance is procured because uniform pressure is transmitted to the entire upper and lower ridges by means of the balance of stress on the dentures. These dentures are balanced in the sense that the distribution of force is evenly divided. In natural dentures this stress is felt solely on the teeth in contact and balance (prosthodontia) is not procured except that the teeth in contact have an even distribution of stress; hence the name equilibrated stress in referring to the so-called balance of occlusion in natural dentures. This same idea is observed in all movements of the mandible. With this thought in mind, in considering the various movements of the mandible, the attainment of equilibrated stress rather than balanced occlusion will be considered.

It is necessary to understand that there are two stresses or loadings to be encountered in the mouth, a vertical loading or stress and horizontal loading or stress. These stresses are normal physiologic stresses which are readily accommodated by the investing tissues. But when this stress is excessive, overloading takes place, and there is the development of vertical overstress or overloading, or horizontal overstress or overloading. Inflammations and symptoms of a destructive character may follow these abnormal conditions. Another important consideration is the fact that there are several conditions in the mouth which must be reviewed in order to understand the mechanics involved in the mechanical disharmony and what biologic responses may be expected.

"The production of overstress in the pericementum (4) is dependent on a disturbed relationship between the applied occlusal stress and resisting periodontal stress; one or the other of these factors must be abnormal and it follows as a complication that both factors may be abnormal, with the result that pathological changes are accelerated and the tooth, as a rule soon lost.

On this distinct basis, according to the mode of origin, traumatogenic occlusions may be classified into the following three groups.

(1) PRIMARY—Abnormal occlusal factor with normal periodontal factor; the magnitude of the original force is so excessive, or the manner in which it is delivered is such that overstress is produced in the pericementum.

(a) Cusp interference in unbalanced occlusion. (b) Excessive wear. (c) Faulty restorations. (d) Dental caries. (e) Uneven wear. (f) Lack of occlusal vents. (g) Displacement following extraction. (h) Unaccustomed mastication of extremely hard foods, holding of pipe-stems, cigarette holders, biting of thread.

(2) SECONDARY—Normal occlusal factor with abnormal periodontal factor; the magnitude of the occlusal force may be normal, but due to inadequate periodontal support, overstress is produced.

(a) Simple border atrophy of the process.

(b) Simplex periodontitis.

(c) Rarefying pericementitis fibrosa.

(3) COMBINED—Abnormality in both occlusal and periodontal factors; when from a traumatic occlusion or other cause, a tooth assumes a position where it receives a still greater occlusal force, due to the already weakened periodontal support, mechanically and frequently pathologically, a double damaging situation is created and unless corrected the tooth is soon lost.

(a) Axial extrusion.

(b) Lateral drifting.

(c) Over-eruption."

The pathologic changes which follow mechanical disharmony may be both subjective and objective, being observed in the form of pain or mobility of the teeth, and more frequently in disturbances in circulation, which are followed by definite pathologic changes. Roentgenograms often depict, too, the various changes which result from traumatogenic occlusion.

With these facts firmly established, and demonstrated by thousands of cases in practice, it becomes imperative to study the means by which health can be procured and maintained in the mouth. Function establishes structure, and by establishing the maximum amount of function in the mouth, health can be maintained. Maximum function may be established by equalizing the stresses on the teeth in the various excursions of the mandible. Fundamental principles must be agreed upon before such procedure can be adopted.

Before attempting to distribute pressures on teeth evenly, a thorough examination of the habits of occlusion of the individual case must be made, (i. e. whether the patient ever has incised food in true protrusive movement or whether this act is accomplished by a shearing stress, etc.). It is important to examine the shape of the teeth and interdigitation of cusps by means of study models and to record all systemic and local findings on a suitable chart. A complete roentgenogram series together with the clinical findings may assist in determining the extent of pathology resulting from the mechanical disharmony. Photographs of the patient will be of great assistance in keeping record of the progress of the case. The color of the gingivae and deviations from the normal should also be noted. This having been done, it is advisable to determine by what means the best results may be accomplished, that is, whether orthodontia, restorations, grinding or combinations of these would be advisable. The method decided upon, we proceed to coördinate the occlusion. Before explaining the

procedure in obtaining equalized stress, it is advisable to compare occlusion in natural and artificial dentures.

### *Centric Occlusion*

| ARTIFICIAL DENTURES | NATURAL DENTURES |
|---|---|
| (1) Centric occlusion must be determined. | (1) Centric occlusion is present, but can be corrected. |
| (2) Simultaneous contact of the opposing teeth should be procured. | (2) Simultaneous contact of the opposing teeth must be procured.<br>(a) Teeth causing food impactions should be relieved. |
| (3) Even distribution of stresses is required. | (3) Even distribution of stresses is required. |
| (4) The lingual cusps of the upper posterior teeth and the buccal cusps of the lowers rest in the central fossae of opposing teeth. | (4) Sulci or fossae should be deepened or cusps relieved. The normal relationship as observed in artificial teeth is not always present. |
| | (5) Correction of centric should be accomplished with no closure of the maxillo-mandibular relation. |

In considering natural dentures in centric relation, care must be taken to observe the proximal contacts of teeth, to see if they are properly located; which teeth are absent and need replacement; whether the marginal ridges and grooves are properly constructed; whether there is contiguity of the teeth in the arch; whether there is uneven wear, etc. These conditions must be carefully observed in order to prevent the development of food impactions. Plunger cusps, both on lingual and buccal surfaces, which might force food in between the teeth are often observed. By grinding, food impactions which have developed as a result of malrelationships existing between the teeth in one jaw, or which may be due to abnormal relationships of teeth of opposite jaws, can be eliminated. The next step in grinding in centric occlusion is to request the patient to close the jaw slowly to determine which tooth or teeth are in premature contact. Roentgenograms, palpation, ocular examination, and study models are excellent aids in determining where overloading in the centric position is taking place. Study models are of great assistance and should be mounted on an articulator in such manner that the articulator will correspond with the patient's movements. Often it is difficult to observe premature contacts in centric as patients have a tendency to glide over the offending teeth and give incorrect registrations. However, if the proper centric relationship between maxilla and mandible is obtained, and transferred to the articulator, the teeth exhibiting traumatogenic occlusion can be observed. The tooth or teeth are noted and comparison is made with the mouth. The patient can locate the tooth by closing slowly and lightly pointing out the tooth which feels higher. (The patient should cease closing as soon as the slightest contact is made.) One should never depend solely upon the patient for information of this nature, but should check this knowledge

with the methods previously mentioned. Patients may feel excess pressure in a certain area, yet point to the wrong tooth. With articulating paper acting as a guide, the portion of the teeth or tooth in premature contact will be marked.

The next step is most important. It must be determined whether the sulcus will be deepened or the cusp in contact with it, will be relieved. The following factors must be taken into consideration.

(1) If the cusp of the lower tooth is in excessive stress in centric occlusion, but in harmony in the lateral excursions, it is logical to deepen the sulcus of the upper tooth. Should the cusp be relieved in this condition, the lateral relation might be disturbed.

(2) If the cusp exhibits overloading in both centric and lateral excursions, it is advisable to grind the portion of the cusp of the lower tooth which is in abnormal stress. This does not mean that the tooth is disoccluded.

(3) If there is premature contact in centric relation and no contact in lateral excursions (both working and balancing sides), the sulcus of the opposing tooth should be deepened. If the cusp of the lower tooth were to be ground, a wider separation between the teeth in the lateral excursion of the jaw would be created.

After these corrections, the pressure will feel equalized over the mouth and simultaneous stress will be observed. Once centric occlusion is established, grinding of the teeth in other excursions should be so accomplished that this relationship will not be disturbed.

Should the lower incisors exhibit no interference in various excursions of the mandible, and cause no strain on the upper teeth in centric relation, they should be allowed to remain in contact. It must be remembered that teeth which have been in contact, and then disoccluded, may again come into contact. (See exceptions under protrusive.) Teeth which are in traumatogenic occlusion are often disoccluded in order to give physiologic rest to the tissues. Should they come into contact later, the teeth will be better enabled to assume the normal stresses which they are expected to bear, as the periodontal tissues will have been rested. Often, too many teeth are brought out of occlusion, unnecessarily, thus complicating the procedure in grinding. When conditions as have been just described in reference to anterior teeth are met the eccentric movement from centric towards protrusive must also be considered. Should the teeth be in traumatogenic occlusion in both the eccentric protrusive and centric, the following procedure should be instituted:

(1) First, relieve the plane of the upper tooth from centric to protrusive so that there will be ease of movement with no tripping.

(2) Disocclude by grinding the lower teeth to give physiologic rest. Should the lower teeth later come into contact there will be no interference in the eccentric protrusive.

## *Protrusive Occlusion*

| ARTIFICIAL DENTURES | NATURAL DENTURES |
|---|---|
| (1) Ease of motion from centric to protrusive with no interference. | (1) Ease of motion from centric to protrusive with no intereference. |
| (a) The lower anterior teeth shall glide over the lingual surfaces of the upper teeth with such ease that the displacement of the denture posteriorly shall not take place. | (a) There should be no displacement of the anterior teeth in their *sockets* as the lower teeth glide over the lingual surfaces of the upper teeth. |
| (b) Cusps of teeth interfering in this movement should be adjusted. | (b) It is important to note whether the cusps of the bicuspid or molar teeth interfere with this movement. The interfering cusps usually are on teeth that have been tipped either buccally or lingually until one cusp or another has been raised out of position. |
| (2) At least three point contact is advisable. When this is procured, the dentures are in balance. | (2) Establish equal distribution of stresses on anterior teeth from cuspid to cuspid, if possible. |
| (3) To establish balance of the dentures, posterior contact must be obtained. | (3) Try to procure posterior contact, if possible. |

The subject of protrusive relation has long been discussed in great detail in view of the many conditions which are encountered in this position of the mandible. More time should be spent in studying the occlusal habits of the patient, in noting whether his incisive action is produced by a shearing force or by the contact of the incisive surface of the upper with the incisive surface of the lower. If the patient possesses no protrusive relation, it might be wise to give him function in this position, thus permitting a movement of the mandible which had not existed previously. Often it may be advisable to leave well enough alone. Incisive action might be ascertained if on examination the incisal edges are found to be worn. In severe overbite cases, it may be advisable to advocate orthodontic treatment or to use artifical restorations or inlays to raise the bite.

As the mandible is brought forward in protrusive movement, and some cusp interferes, this offending cusp should be located and relieved, care being taken that, if possible, centric relation is not disturbed. Grinding of the teeth is accomplished by the use of a number 5 or 64 Chayes or the Popper stones, and should be done in such manner that the principle of cusp form will not be destroyed. Should there be displacement of the anterior teeth in their sockets during this movement, the angle of inclination should be changed. This should be done by grinding the lingual surfaces of the upper anterior teeth. These surfaces (contact of labial of the lower against lingual of the upper) should present inclinations which have essentially the same relationships as the cusp surfaces of the posterior teeth. If these inclined planes are out of harmony with the posterior inclined planes or with each other, excess lateral overstress will develop.

In the course of grinding in this relationship, it is suggested that the grinding be confined to the area from centric contact to along the lingual surface of the upper teeth. If the portion of the lingual surface of the upper tooth which established contact in centric, or the lower tooth is ground, the teeth may be disoccluded in centric relation, and thus a condition not intended for these teeth is created. At some future time, the lower tooth may move upward in centric and the original condition will have been reestablished. By confining the grinding from the point of centric contact along the plane of the lingual surface of the upper tooth, ease of movement may be obtained without causing tripping in protrusive thrust and this will also prevent the disocclusion of these teeth when they assume centric position (Note exceptions under centric).

In severe overbite cases when contact in centric is made between almost all of the labial enamel surface of the lower with the lingual surface of the upper, it may be advisable to compromise. In this relationship, one can change the plane of the lower tooth by grinding from the lowest point of centric contact on the lower, towards the incisal edge. In view of the fact that the *incisal edge* of the lower makes no contact with the lingual of the upper in this particular case, one can remove almost ⅛ of an inch of tooth structure inciso-gingivally if necessary, on the lower without interfering with centric occlusion, this relation having been established by the contact of the labial surface of lower with the lingual surface of the upper. It is understood that the procedure is only followed in these cases of severe overbite.

The esthetic should be observed as much as possible during the entire process of grinding, so long as it does not interfere with the proper functioning of the jaws. Occasionally, grinding for esthetic relation in one movement of the jaw may cause the development of non-occlusion in another excursion.

It is not necessary to procure posterior contact in natural dentures, if grinding will tend to make anterior teeth appear unsightly or extremely sensitive. Of course, the more teeth in contact during the protrusive position, the more the distribution of equalized stress. It should be remembered, however, that we are not dealing with full dentures and there is not the need of concern over a displacement of full upper denture, through lack of posterior contact. For this reason, in some cases, it may be advisable to distribute the stresses in such manner that the central and lateral incisors, only, are in simultaneous contact in the protrusive relation. When grinding in protrusive, to procure contact of the incisal edges of upper and lower anterior teeth, it is advisable to confine the grinding to the incisal edges of the upper teeth. If the incisal edges of the lower teeth are ground, non-occlusion in centric relation may occur. If grinding is confined to the edges of the upper teeth, there will be no interference in the other excursions of the jaw.

Care must be taken to be certain that the patient is giving a true protrusive movement. The median line can act as a guide to the operator.

## *The Lateral Movement*

| ARTIFICIAL DENTURES | NATURAL DENTURE |
|---|---|
| It is an established fact that working and balancing sides are important factors in the establishment or stability and function of the denture. | One must correct whatever abnormalities of wear which nature has developed regardless of the cause. It is advisable, if possible, to procure contact on working and balancing sides. However, the balancing side in some instances cannot be procured. |

The cusps which had maintained contact with opposing tooth surfaces in centric position, should also maintain contact in lateral movements of the jaw. The mastication of food depends upon two factors: the sharpness of the cusps and inclines of the planes and the points of the cusps functioning on the inclines. Often in the process of correction, cusps are ground and their sharpness removed, never restored, and the result is discomfort and inability to masticate food properly.

During the lateral movement of the jaw, we think of the working and balancing sides. In this movement of the jaw, the lower buccal cusps on the working side glide along the planes of the buccal cusps of the upper teeth in a scissors-like fashion, no tripping, nor displacement of the upper teeth taking place. During this movement, there should be contact of the teeth on the other side, called the balancing side. Should excessive stresses be noted, it may become necessary to reduce, and in some instances, eliminate the inclined planes.

The modus operandi for relief in the lateral movements of the jaw is the same as in other positions; all means of determining excessive pressure, roentgenograms, palpation, ocular examination, study models, patient's response, etc., are employed. With articulating paper as a guide, the patient is instructed to close the jaws in centric relation and then to move the jaws laterally. The tips of the fingers can be placed on the teeth on the working side to note whether any displacement takes place. The following procedures should be carried out:

(1) The anterior teeth may be engaged during this movement. They should be relieved by grinding along the planes of the upper anterior teeth, so that centric will not be disturbed.

(2) The cuspid teeth often interfere during the lateral movement of the jaw, thus preventing contact with other teeth on the working side. The cuspid teeth should be in contact throughout centric and eccentric positions. In the event that the cuspid tooth interferes with correct lateral excursions, it should be ground from the point of centric contact along the path of the upper tooth. Should the lower cuspid be ground, it would take the lower cuspid out of contact in centric and will not again be in contact until the extreme excursive movement is reached. An overload is also placed on the remaining teeth in the arch. If the lower cuspid is ground it may exfoliate at some future time until

contact in centric relation is again established, and thus re-establish the original premature contact in lateral movement.

(3) The lower teeth now glide along the planes of the cuspid, bicuspid and molar teeth with ease, with no tripping in the excursive movement. If during this lateral movement, it should be found that a tooth moves in its socket beyond what is considered normal, the presence of traumatogenic occlusion may be considered established. The traumatized teeth can be located, the articulating paper acting as a guide. The planes of the upper teeth are ground from the point of contact in centric along the buccal inclines of these teeth. The surfaces of the teeth causing this undue pressure are relieved until they no longer interfere with the lateral movement. Teeth should glide over each other as do the blades of shears during the act of cutting.

(4) Often the lingual cusps interfere, and it is essential to confine grinding to the planes of the lingual cusps of lowers rather than the cusp of the upper. Grinding the cusp of the upper would interfere with the centric occlusion, in view of the fact that in centric occlusion the cusps of the upper posterior teeth and the buccal cusps of the lowers rest in the central fossae of the opposing teeth.

(5) The balancing sides must also be taken into consideration when the lateral excursions are considered. Here, two considerations determine our procedure: (a) The health of the teeth on the working side. If the teeth on the working side are loose, it becomes imperative to procure contact on the balancing side in order to relieve the strain of the loose teeth on the working side. (b) Should the teeth on the working side be strong and in good relation, and should further grinding to procure balance on the balancing side tend to imperil the form, sharpness or sensitiveness of the teeth on the working side, it is advisable to leave well enough alone. After, all, we need not fear for the displacement of the dentures as in full dentures. This lack of balance will cause a disturbance in equilibrium and a failure of the artificial denture to function well, whereas in natural teeth, such concern is not necessary. However, the more teeth in contact, the better the distribution of forces.

(6) Often, more pressure is felt on the balancing side than on the working side and here is a problem that is rather serious. The contact on the balancing side is made by the gliding of the inclined plane of the buccal cusp of the lower tooth along the lingual cusp of the upper tooth. Fortunately, in most instances, it is rather the disto-buccal groove of the lower which makes contact. Should the lingual cusp of the upper be ground, then non-occlusion will result in the centric position and thus place a greater burden on the remaining portion of the tooth. By widening the disto-buccal groove on the lower tooth, the excess pressure can be relieved without causing an upsetting of the centric relation. Should this relationship not exist, the models should be carefully examined and

grinding be confined to the portion of the teeth which would least interfere with centric relation.

The so-called balance in the lateral movements of the jaw, should, when completed, give a normal contact on the working sides and balanced occlusion on the balancing sides. Tripping by the cusps in the oscillating movements of the jaw has caused good bridgework to loosen. This excessive pressure has produced mobility in otherwise healthy teeth.

### *Final Check-Up*

After all the grinding has been completed, it is necessary to check back in order to determine whether coördination is present. When harmony in these movements has been obtained, and the minimum of tooth displacement has resulted, the coördination of the occlusion and equal distribution of stresses may be considered established.

### *Suggestions*

(1) All suggestions given for the relief of traumatogenic occlusion are not followed in every case. Sometimes there are too many factors which will interfere with the attainment of the ideal. Each case presents certain obstacles which must be overcome, and can only be accomplished if fundamental principles of occlusion are clearly understood. Common sense should always be the guide.

(2) When palpating for displacement of teeth, use the index finger of the left hand, which seems to be more sensitive than the right.

(3) To remove additional high spots, or rough surfaces, after grinding, incorporate carborundum paste into softened chewing gum. This should be used only for smoothing surfaces and not for reducing premature contacts.

(4) Even though only one or two teeth may be affected, there is the possibility that others should be relieved to accomplish the desired results.

(5) At times patients are unable to move the mandible laterally. This is usually a result of an abnormal overbite or close interlocking of cusps.

(6) The teeth should be polished with smooth cuttlefish discs after grinding.

(7) Be certain that patients feel comfortable after each treatment. It is suggested while grinding that the tooth be held tightly by thumb and index finger. It lessens vibrations of engine.

(8) In the course of the relief of one side during lateral movement, observe that the cusps of the other side are free from interference to the proper movement.

(9) Where the form of the tooth or density of tooth structure, or an artificial substitute, does not allow for grinding, use the opposing tooth. However, where this is done, the operator must be acquainted with the possible con-

sequences. If the offending member is grossly out of harmony, it may be necessary to extract, or, in the case of a restoration, to replace it.

(10) Do not heat the stone; cool it by spraying with water. While grinding do not keep the stone on one spot for any length of time.

(11) Be careful to observe and to correct factors which might cause the development of food impactions.

(12) At all times restore the tooth form.

(13) a—If a sensitive spot or so-called erosion cavity is encountered, dry into the area 40% formalin. Dry the tooth well and apply formalin to the area on a small pellet of cotton. This is followed by the application of hot air, and relief of pain results. Another method is the rubbing of formalin into the area by heating an orange-wood point immersed in formalin over a flame and rubbing it into the sensitive location. Care must be taken to protect the soft tissues. b—A saturated solution of potassium carbonate in glycerin (200 gr. of potassium carbonate to 1 oz. of heated glycerin) may be applied to the sensitive spot by an orange-wood point for about two minutes. There need be no concern over the soft tissues as the drug has no caustic action.

(14) If crowns or bridges interfere with procuring coördination, they should be removed and new ones placed.

(15) By all means be certain that your diagnosis and prognosis are correct before commencing to grind.

I wish to express my thanks to Professor Clyde H. Schuyler of New York University College of Dentistry for his valued suggestions during the preparation of this paper.

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# Incidents of Office Practice[1]

## *Bismuth Stomatitis*

*By* WILLA YERETSKY, *D.D.S.*
Grand Rapids, Michigan

In recent years preparations of bismuth have come into favor as substitutes for mercury in the treatment of syphilis and other diseases. Bismuth salicylate is the form more commonly used at present and is generally given in an oil suspension intra-muscularly. Because of the possible cumulative effect of bismuth as well as a sensitiveness of the patient to the drug, a severe stomatitis sometimes results. In the earlier days of the use of bismuth, the percentage of stomatitis cases resulting ran as high as 15 to 20%. Now that the bismuth preparations are more refined and the treatments spaced further apart, only about one per cent of cases treated result in a stomatitis. This applies chiefly to those cases treated intra-muscularly and very rarely to those where bismuth is administered by mouth for other reasons such as X-ray or gastro-intestinal disorders.

Bismuth stomatitis is similar to mercury stomatitis in its effect upon the gingival tissue but differs somewhat in appearance. In the case of bismuth there is a more prominent bluish or blackish line. There is also a fetid odor which sometimes precedes the pigmentation. The onset of the stomatitis is very sudden. To produce this stomatitis it is necessary for an abrasion to occur along the gum margin. This may be due to calcareous deposits or to injury of a gum flap over a partially erupted third molar—"Bacterial flora then gain a foothold and form hydrogen sulfid for the formation of bismuth sulfid deposits upon which further necrosis depends." (Stokes).

These cases are very amenable to treatment. It is of the utmost importance that a thorough scaling and polishing be done as quickly as possible. Since saprophytic flora thrive on the detritus in such mouths, it is often advisable to use anti-Vincent's disease treatment. I have found irrigation with a 5% chromic acid solution very useful in these cases with a simple mouthwash of normal saline solution with a like amount of sodium bicarbonate for home use. A mouthwash recommended by Stokes is one of liquor antisepticus alkalinus containing ¼% zinc chlorid. The teeth should be vigorously brushed in order to keep them perfectly clean. Acid foods should be avoided for the duration of the stomatitis. Under this regime plus the use of sodium thiosulfate by the physician such cases will usually clear up in about a week.

## *Prescribing For Patients in Periodontia*

*By* EDWARD L. BALL, D.D.S.
Cincinnati, Ohio

Recently an incident occurred that brought to my attention a seemingly lost art in the daily practice of dentistry, i. e., the art of prescribing to patients, either pre-operative or post-operative.

The following case report speaks for itself.

After surgically treating seven lower anterior teeth, the nervous patient was given *oral* instructions as to home care. Incidentally, this was an out-of-town patient.

Five days later the following letter was received:

My dear Doctor:

"We forgot the mouth wash after all! As you may imagine, I have had a rotten week, both literally and figuratively speaking. Had quite a temperature by the time I got home Friday evening, but better Saturday so I thought nothing of that, but Sunday afternoon the temperature came up again, and persisted through Monday morning. Went to Dr. Doe, who swabbed the

[1]Read at the twentieth annual meeting, American Academy of Periodontology, August 3, 1933, Chicago.

festering gums and gave me a mouth wash. Temperature normal since, gums better; my eye has given a little trouble, but not so bad as I expected; I have an annoying bronchitis and my sinuses on the left side feel sore; I've had a very slight touch of lumbago.

"I wonder if you still will want to see me Saturday? I'm afraid there hasn't been a great deal of healing."

Very truly yours,

---

How much distress I could have avoided if I had written the following instructions:

1—Salt water mouth wash—½ tb. to ½ gl. of warm water every two hours.
2—Cold applications.
3—Aspirin, grs. 5 every four hours Saturday and Sunday.
4—Saline carthartic tonight.

Those of us who are interested in periodontia have given a great deal of thought and care to the technique of toothbrushing, with excellent results.

So often, before we are half through with our brushing instructions the patient will ask the following questions: "Doctor what tooth paste shall I use? Which one is best for my mouth? I believe I could go to a dozen different dentists in the Doctors Building and get a dozen different suggestions as to what toothpaste or mouthwash to use." What is my stuttering answer? For this patient, I immediately write a prescription stressing its importance.

Successful physicians are as a rule clever prescription writers. Psychologically, a prescription has a marvelous effect. Then, why have the dentists failed to take advantage of this psychic reaction? A check up of fifteen different drug stores gave me the information that only two dental prescriptions had been received in the past six months. It is not my intention to suggest writing prescriptions for all patients, however, writing instructions for all patients is a necessity both for their comfort and our protection. Many proprietary drugs can be safely prescribed and the Council of Dental Therapeutics will gladly send you an accepted list of therapeutic agents. Cases present themselves now and then where warm water is the solution of choice. Often the patient by using strong mouth washes has not given the tender tissues a chance to heal. Then, two main points to consider in prescribing a mouthwash are, efficiency and safety to tissues.

The surgical technique for the elimination of the pyorrhetic pocket has been so generally accepted that both pre-operative and post-operative instruction to the patient are of the utmost importance. The patient's physician should be consulted before the operation and after, if you are worried at any time. The following considerations are important in any surgery of the mouth and jaws:

PRE-OPERATIVE:

After a general prophylaxis, the local treatment advocated by Dr. Vastine is started. The patient is instructed as to the proper toothbrush technique and hydrogen peroxide or sodium perborate mouthwash is prescribed.

POST-OPERATIVE:

The following post-operative instructions may be printed in pamphlet form and given to the patient: They refer to rest, laxatives, diet, mouth washes, pain, fever, teeth and gums, hemorrhage, loss of weight.

*Rest:* For the first three days after operation, the patient should rest as much as possible. Rest in bed is a particularly necessary precaution for patients who have had rheumatism. Those whose general health is not affected need not adhere strictly to this rule; in fact, there are some who return to work the second or third day following the operation.

*Laxatives:* On the second day following operation, any laxative to which the patient is accustomed should be taken. Pluto water, aromatic cascara, or Epsom salts may be suggested. If there is not sufficient action from the use of a laxative, an enema of a tablespoonful of baking soda in a pint of water should be given.

*Diet:* On the day of the operation, cold fluid diet should be taken. Beginning on the second day and continuing until the surgical dressing is removed or tissues are comfortable, a week's program of soft diets is given the patient. Encourage the drinking of water and for a short period one-half to one teaspoonful of baking soda to each glass is beneficial. If preferred, Vichy water may be taken instead.

*Mouthwashes:* It is not advisable to

rinse the mouth more than four times a day. Too frequent rinsing has a tendency in some cases to cause a slight hemorrhage or loosen the dressing. Baking soda or salt, a teaspoonful to a glass of warm water, is recommended. If there is pain in the mouth or throat, pulverize one aspirin tablet in three tablespoonfuls of water and use as a gargle or mouthwash.

*Pain:* Some patients have great relief from the use of an ice bag or a cold wet pack. This pack is made by loosely wringing a towel which has been dipped in ice water. It is then folded and placed at different intervals to the side of the jaw operated upon. After applying the towel, cover it with a dry towel or oil silk to retain the moisture. The patient is then given a Barbital or Amytal Compound to assure rest the first night. Aspirin grains 5, usually alleviates any pain.

*Fever:* A slight rise of temperature often takes place after the operation. The fever is usually controlled by sponging the body with luke warm water or by giving a warm bath. After a good bowel movement, the fever usually disappears. If the temperature remains high, Aspirin grains 5 is given every three hours.

*Teeth and Gums:* Patients must not brush the teeth until after the surgical dressing is removed but should rinse the mouth gently with warm salt water. A soft brush is given as soon as it is practical. It is important that the dressing stays in position for at least seven days.

*Hemorrhage*: Fortunately, this complication is of rare occurrence. When it does take place, the patient is to remain in bed with the head well elevated, using three or four pillows. A slight bleeding is easily controlled by, not rinsing the mouth, remaining absolutely quiet, and not becoming excited. Profuse bleeding should not be allowed to continue without communicating with the dentist's office. Merrill's Oral Fibrogen is suggested in such cases.

*Loss of Weight:* During the first week following surgical treatment, there is usually a loss of weight, which is soon regained.

May I urge the use of the following simple prescriptions and instructions to patients which have been successfully used in many offices:

WRITTEN INSTRUCTIONS TO PATIENTS:

*No. 1. Following Surgery*

℞

1. Ice bag to jaws
2. Cold salt water mouth wash today
3. Warm salt water mouth wash after today
4. Aspirin for pain
5. Cathartic
6. Drink plenty of water

*No. 2. For Acute Vincent's Infection.*

℞

1. Peroxide of hydrogen (full strength) mouth wash every two hours
2. Four glasses of orange juice daily.
3. Aspirin for pain
4. Cathartic
5. *Rest* in *bed*
6. Boil dishes
7. No smoking

PRESCRIPTIONS:

*No. 1. Vincent's Infection.*

℞

| | |
|---|---|
| Hydrogen dioxide aquae | 8 oz. |
| Mercury bichloride | 2 grs. |

*Sig*: Two teaspoonsful in ½ glass of warm water every hour. Use as a mouth wash.

Note: Some cases alternate with the following:

℞

| | |
|---|---|
| Potassium permanganate | 15 grs. |
| Distilled water | 6 ozs. |

*Sig*: Tablespoonful in a ¼ glass of warm water. Use as a mouth wash.

Note: From a paper on Vincent's Infection by: Arthur H. Merritt, D.D.S.

*No. 2. Tooth Powder for Daily Use.*

℞

| | |
|---|---|
| Calcium carbonate precip. | 8 drs. |
| Sodium perborate | 4 drs. |
| Powdered castile soap U. S. P. | 40 grs. |
| Saccharin | 1½ grs. |
| Oil of wintergreeen | 15 m. |
| Oil of peppermint | 2 m. |

*Sig:* Use on toothbrush night and morning in brushing gums and teeth.

(Prescription from "Periodontal Diseases", by: Arthur H. Merritt, D.D.S.

*No. 3. For Sore Throat and Painful Mucous Membranes.*

℞

| | |
|---|---|
| Aspirin | 7 grs. |
| Phenacetin | 6 grs. |
| Codeine phosphate | ¼ gr. |

M. Ft. pulv. No. 1

D. T. D. pulv. No. 12
*Sig*: Dust on gums or throat ten minutes before eating.
Note: Prescriber must have narcotic license.
*No. 4. Hypnotics.*
℞
Barbital sodium 5 grs.
*Sig*: One or two tablets before retiring.
or
℞
Sodium amytal 3 grs.
(Lilly)
*Sig*: One capsule before retiring.
*No. 5. For Swelling or Cellulitis.*
℞
Epsom salts } saturated solution.
Sodium chloride }
*Sig*: Use in tepid water. Apply externally. Keep dressings continually moist.
or
℞
Epsom salts (saturated solution)
*Sig*: use as directed.
Apply externally using hot, moist dressings continually.
*No. 6. For "Bleeders".*
℞
Oral Fibrogen (Merrill)
*Sig*: Empty vial in a large glass of ice water and give to patient on empty stomach. Have patient lie on right side, right arm extended above head.
*No. 7. For Hospital Cases.*
℞
P. P. O.
1. Sodium amytal 3 grs.....8 P. M.
2. No breakfast
3. Sodium amytal 3 grs.....8 A. M.

Note: Write all instructions on daily chart or order book.

*Post-Operative Instructions.*
℞
1. Ice bag to jaws
2. Codeine sulphate ½ gr.
(H) P. R. N.
3. Cold salt water mouth wash today Q. *i. d.*
4. Warm salt water mouth wash after today. Q. 2h.
5. Liquid diet.

℞
Note: Prescriber must have narcotic license. For additional information on accepted remedies write to the Council on Dental Therapeutics, of the American Dental Association.

In conclusion, may I quote from a paper written by Dr. Arthur P. Luff in the British Medical Journal.

"The gauge by which your success in practice will be mainly measured and the esteem in which to a great extent you will be held by your patients, will depend upon the success you attain in the treatment and cure of what may superficially appear to be trifling ailments, but by the individual sufferers considered important and therefore deserving the careful attention and the skilled therapeutic resources of the medical man."

Prescriptions from Prinz's Dental Formulary, Merritt's Clinical Periodontia, Dr. Frank Kaisers' writings, Drs. Mithoefer & Bryan. Pamphlet on instructions to patients following tonsillectomy.

## *An Embarrassing Experience Following Gum Resection*

*By* JESSE L. WILLIAMS, D. D. S.
Jacksonville, Fla.

Mrs. D., age about fifty presented for treatment of periodontal involvement on November 17, 1932, at which time a general mouth X-ray examination was made.

There were some minor periodontal pockets scattered about her mouth, and also quite a deep pocket involving the upper left second molar, the first molar having been lost many years previously. This pocket involved particularly the mesio-buccal root extending slightly into the bifurcation of the buccal roots.

On November 20th a bacteriological examination was made of a smear from her mouth which showed staphylococci and bacilli to be present but no Vincent's organisms were found. The patient's dental history showed nothing unusual as to her reaction to dental treatment.

The conservative method of treatment was used on all her teeth except the upper left second and third molars. A complete gum resection was used on these teeth on December 8th.

The usual cement pack was placed in and about these teeth and the patient dismissed. On the 10th she returned to the office for the usual check-up and at that time a mild ulcerative stomatitis was present. The patient's mouth was sprayed with a solution of menthol and boric acid with the Dunlop Spray.

On the following day she called me requesting that I come to see her, that she was in considerable pain, and discomfort. On examining her mouth I found quite an aggravated ulcerative stomatitis involving the buccal tissue adjacent to the pack, and also the superior left side of her tongue showing the same ulcerative condition with a beginning slough in both these regions.

The patient's temperature was 103; the left side of her face was considerably swollen and the glands about her neck on this side were considerably enlarged. The tissue immediately underneath the pack appeared healthy, that is, where the tissue had been resected. I immedaitely removed the pack, applied cold applications to the patient's face to reduce the swelling, swabbed out the mouth with metaphen—1-2500, advised a laxative and applied zinc chloride 10% to the lesions.

On the following day I visited the patient again. Additional areas showed evidence of the spread of the ulcerative stomatitis and I called in a nose and throat specialist for consultation.

He reported nothing except as mentioned above, saying that he felt perhaps the oil of cloves in the pack had been responsible. I saw the patient every day for the three following days but saw no great improvement.

I then asked her to see a competent diagnostician who made a check up reporting nothing except as above mentioned, an extremely aggravated case of ulcerative stomatitis.

The patient then recalled an unpleasant experience following a facial treatment at a beauty parlor while on a visit to California some years previous. She reported that at that time her eyes and face were badly swollen.

I sent the bottle of liquid used to mix the pack to the manufacturers for analysis. They reported it as being free from impurities and all right in every way.

The conclusions we arrived at were, that we were dealing with a case of unusual susceptibility to certain drugs contained in the liquid, or possibly to some obscure infection in her mouth at the time of operation, which was not discoverable bacteriologically.

## [*Case* I.] *Vincent's Infection*

*By* SIDNEY SORRIN, D. D. S.
New York, N. Y.

In the last few years, there has been a growing tendency in the medical field to treat Vincent's infection with intra-venous injections of neo-salvarsan, combined with the use of an oxygen-liberating mouthwash. The result of this therapy has caused a subsidence of the symptoms in some cases, but at the same time a subacute or chronic condition has developed. This procedure in the treatment of Vincent's infection should be discouraged.

Recently, Dr. N. Rosenthal, well known hematologist, connected with Mt. Sinai Hospital in New York, in a talk before the Pathodontia Section of the First District Dental Society suggested that the practice of intra-venous injections should be discontinued because it did not remove the cause of disease and made the patient most uncomfortable.

The following case report is therefore cited: Mr. G., age 23, white, healthy, habits good, presented with an acute Vincent's infection. (Fig. 1)

*History as recited by patient,*—Sept. 22, 1932.

"About the seventh of August, I first noticed an uncomfortable feeling in the mouth. My tongue was badly affected and I was unable to eat. I was troubled with grinding of the teeth during sleep and by bleeding gums.

"On August 11th I went to see a throat specialist as my throat began to be painful. He painted my gums and throat with an aniline dye and told me if it was no better

to call him the following Monday, August 15th. The dye relieved the pain for a short time but over the week-end the condition had become such that I was unable to eat any solid food or to drink any hot or spicy fluids because of the pain in the swollen and lacerated tongue and the swollen gums.

FIG. 1

"I called him on Monday, the 15th, and made an appointment. He gave me an injection of neo-salvarsan intra-venously, and sent me home, where I remained in bed for the balance of the day. After about thirty-six hours I noticed a slight improvement, my tongue clearing up, but my gums still bothering me. I called at his office again the following Thursday and was given another injection of neo-salvarsan and was told to stay in his private hospital for a few hours until the effects of the injection had worn off. This I did, then went home and to bed until the following day. From that time on, I noticed a continued improvement in both my gums and tongue, and had no further injections until about four weeks later when I received another injection which did not produce any improvement. During the entire period of treatment no oral prophylaxis was advised. I went to see my dentist, Dr. E., who treated my gums but said that he would like to have me see a specialist and made an appointment for me with Dr. S., whom I went to see in September."

FIG. 2

## TREATMENT

The dental treatment consisted of a complete radiographic and photographic study with confirmatory bacteriological smears together with accurate charting and the taking of a set of plaster casts. Rough surfaces of the teeth which had caused constant irritation to the tongue were noted. The clinical picture was pathognomonic of Vincent's infection. Patient had been eating in a lunch wagon and was advised against this practice.

Instruction in diet and oral hygiene was given and the patient advised to use a perborate mouthwash. Toothbrush drill was demonstrated and checked, and irritating surfaces of the teeth were corrected. Acri-violet was ap-

plied daily and instrumentation instituted after the third day. Food impaction which developed as a result of mal-relationship between the teeth, was relieved by grinding and the mouth given maximum function by distributing the stresses equally on the teeth in all the movements of the jaw. Marked improvement was noted within two weeks and all symptoms subsided within six weeks. (Fig 2)

It is now six months since completion of the treatment and there has been no recurrence. The tongue which had previously been irritated and raw is now healthy with no signs of infection. There is no more grinding of the teeth during sleep, nor bleeding of the gums.

I regard it as most important to place the mouth in an hygienic condition as quickly as possible, and to emphasize the need of a protective diet. The sooner this is done, the sooner will the Vincent's infection be entirely eliminated.

## [Case II.] *Myelogenous Leukemia*

This case of myelogenous leukemia is presented with the thought in mind of stressing the necessity of a blood examination in treating dental conditions.

*History*: Patient, unmarried woman, age 42, white, suffering from a lowered resistance; referred by family physician to an oral surgeon for the removal of any foci of infection. The lower right first molar was pulpless and the third molar was impacted. These teeth were removed on July 1, 1931, and swelling, pain, etc., were noted the following day. There was an infected socket which was treated for a period of ten days. On July 21, the condition was not improved and inflammation of the gingival tissues was noted. Smears and cultures revealed a mixed infection of the gingivae with a preponderance of Vincent's spirillae. A few days later ulcers developed on the gingivae, tongue and cheek and the patient was referred back to her physician for a blood count. The blood count was normal and the patient was treated with sodium perborate and salvarsan. On July 28, 1931, the blood count revealed a change and the physician reported as follows:

July 28—Haemoglobin 60%. Red cells 3,000,000; white cells 27,000; second salvarsan given; no enlargement of the spleen.

Aug. 1—Haemoglobin 53%. Red cells 2,700,000; white cells 40,000; spleen slightly enlarged; third salvarsan.

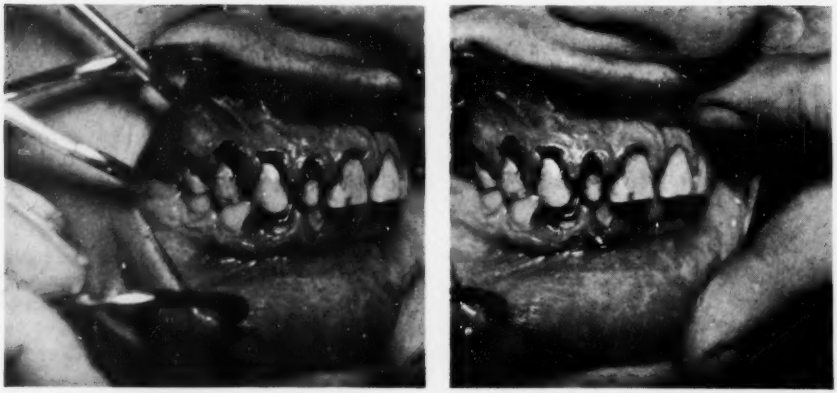
Aug. 7—Haemoglobin 49%. Red cells 2,700,000; white cells 45,450; polymorphonuclears 68%; small lymphocytes 5%; large lymphocytes 5%; myelocytes 22%. Red cells showed marked variation in size and shape and there was marked polychromatophilia. Smear from mouth, positive for Vincent's infection.
Fourth sal-varsan.

Aug. 12—Roentgen treatment given to the spleen and long bones, daily.

Aug. 14—Fifth salvarsan.

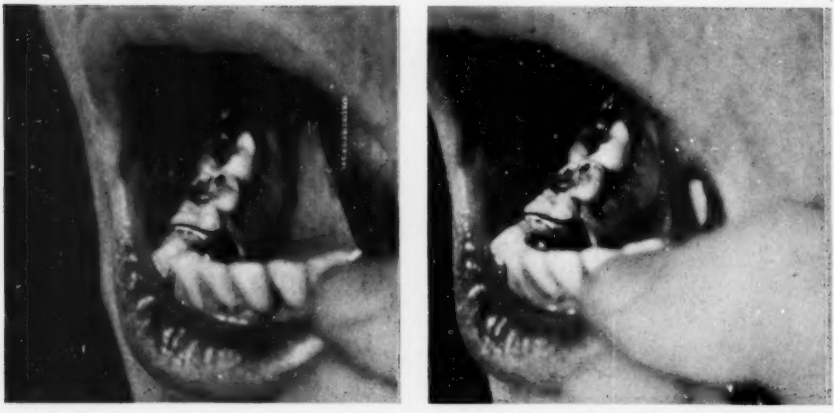
There was a marked anisocytosis and

*Myelogenous Leukemia Labial View*



*Buccal view*

*Upper lingual view*



*Lower lingual view*

poikilocytosis. Numerous nucleated red cells—both macroblasts and normoblasts and many macrocytes were present.

Sept. 8—X-ray series finished.

Sept. 19—Haemoglobin 50%. Red cells 2,790,000; white cells 56,300; neut. polymorphonuclears 71%; lymphocytes 2%; transition 1%; myelocytes 26%.

Oct. 21—Haemoglobin 55%. Red cells 3,970,000; white cells 89,200; neut. polymorphonuclears 61%; lymphocytes 2%; myelocytes 37%.
Many nucleated red cells seen.

Nov. 17—Haemoglobin 54%. Red cells 3,570,000; white cells 71,200; neut. polymorphonuclears 54%; lymphocytes 4%; myelocytes 42%.
Numerous nucleated red cells seen.

The use of Roentgen ray over the spleen and long bones resulted in a decrease in the volume of the spleen and an increase in red cells and haemoglobin. The white cells were first materially decreased but again the blood became leukemic. The myelocytes increased in number, and the lymphocytes relatively less in number 1-4%.

The case was referred to me on December 7, 1931, for palliative treatment. I was surprised at the attitude of the patient. She complained of feeling tired, but otherwise well and happy. Her chief concern seemed to be her mouth and her desire to return to work. The mouth conditions had grown progressively worse as the photographs will show. The gingival tissues were proliferated with hemorrhagic and purpuric manifestations.

Mild alkaline mouth washes were suggested and nothing further was heard from patient. Two days later she died while riding in a cab.

This case illustrates the care which should be taken in cases of this nature, where lack of information regarding the case might result in a fatality for which one might be held, even without due cause. May I offer, then, the suggestion that in cases of proliferative gingivitis, or where the etiology of the disease is indefinite, that the patient undergo a complete differential blood count.

* * *

# The National Museum of Dentistry

*By* JOHN T. HANKS, D.D.S.
New York City

The writer had occasion to visit Washington and to seek an interview with the Surgeon General of the Army and at that time visited the National Museum. He found there a dental section of considerable size having a dental officer, Major James B. Mann, in charge. The collections were well arranged and catalogued. They consisted of both anatomical specimens and historical instruments and appliances, housed in a room 18x52 feet. There were also many duplicate anatomical specimens available for study.

Accredited workers have the privilege of study and research, are assigned rooms and given every opportunity to pursue their work. In fact the resources of the whole Museum are at their command, as well as the Surgeon General's Library housed in the same building, consisting of over 500,000 volumes, together with numerous reprints, magazines, etc. There are thousands of slides, sections and gross specimens. The photographic department is most complete.

At the present time plans are prepared for a new building to be erected on the grounds of the Medical Center, where are also to be found Walter Reed Hospital, the Army Medical School, the Army Dental School and allied activities. In the plans for the new library and museum building, the space assigned for the dental section is 134x54 feet, or nearly eight times the size of the present quarters.

The Museum at present has about 8,000 visitors a month. A more complete dental exhibit would be interesting to about 100,000 visitors a year.

What we specifically desire to bring to the attention of your Society at this time is the proposal that your body assume an active share in the plans of the Surgeon General through the formation of a "Registry of Dental and Oral Pathology" along the

lines now laid down by the National Registry of Pathology under the auspices of the National Research Council.

It is proposed that a Registry of Dental Pathology be established to compare with those already functioning at the Army Medical Museum. There will be sections of this registry representing each participating constituent branch of the American Dental Association. This registry will be conducted by a specially chosen commissioned officer of the Dental Corps of the United States Army, who will be known as its Registrar. He will be under the jurisdiction of the Curator of the Museum. A committee will be appointed in each such society to stimulate the use of this registry and to act as consultants when called upon by the Registrar.

We ask that your society endorse this plan for the establishment of a Registry of Oral Pathology and appoint such a committee.

The materials in the registry will be available, to a reasonable extent, for loan to contributors upon approval of the Registrar and consulting committee concerned. In all cases contributors will be accredited for the material used.

Your Society can secure such a quantity and variety of specimens, which may be arranged and housed as to make a national center for the study of all phases of your specialty. There can be gross specimens in such number as to show the various modifications of the different lesions found in the Oral Cavity. Also sections, slides, photo micrographs and photographs can be prepared in endless numbers. Study rooms with every facility can be assigned to members of this Society, who will be accorded all privileges of the Museum and Library to enable them to pursue investigation in their specialty.

The Surgeon General, The Officers and Curator of the Museum and the members of the Museum Committee place themselves at your service.

* * *

# Local Anesthetic Solutions*

*By* LEO SCHUCHARD

Chief, Division of Exodontia, Dental Staff, Children's Hospital, San Francisco

The injection of a foreign solution into the human tissue brings about certain changes. We know considerable about these changes, but very little concerning the principles involved in bringing about these changes.

The human organism is tolerant of many abuses, and it is fairly logical to assume that some of the local anesthesias are abuses. All anesthesia, whether local or general, is an experimental procedure. Unfortunately, however, the object of the experiment cannot always be followed to a satisfactory conclusion. There is no way of telling how much tissue damage has been done, nor how seriously the physiological condition of the organism has been altered. As a rule, many consider the experiment a success when anesthesia has been established and the operation completed, but really the experiment is not finished until the operated tissue and the entire organism is normal again.

In 1928 Guido Fisher published an article in which he applied the principle established by Von Gaza and Brandi (1927). The latter authors found that inflammation of tissue was associated with a local acidosis, and the degree of acidity depended on the acuteness of the inflammatory reaction. They showed that injecting acid solutions in such inflamed regions caused pain, while alkaline solutions with a pH of 8.0 could be injected without pain. Fisher applied this high pH solution (alkaline) in painful mouth infections with marked beneficial results. He then used this principle for local anesthetic solutions and found that the time and the depth of anesthesia were considerably improved. There was no after pain. He stated that anesthesia took place immediately after injection, whereas with acid solutions the acidity first had to be neutralized by the tissues before anesthesia could be established.

About the same time, some investigations were started in Northwestern University Dental School. In 1929 H. T. Dailey and H. C. Benedict reported on the Goldfish

*Syndicated among members of American Dental Editors' Association.

method of testing changes on local anesthetics. This led to a comparative test of procaine and cocaine hydrochlorides, butyn sulphate and procaine borate. As indicated by this method, procaine borate was the most effective agent, butyn sulphate was second, cocaine hydrochloride third, and procaine hydrochloride fourth. Whatever the value of the Goldfish method may be, that is, whether it measures toxicity or anesthetic action, the method may serve for comparing one anesthetic agent with another on a live object.

On investigating the reason for the greater effectiveness of procaine borate, it was found that boric acid is a weak acid as compared with hydrochloric. A solution of the salt of boric acid will be much less acid and will have a higher pH *value.*

Tests of a procaine hydrochloride solution showed a pH around 5.5; of procaine borate a pH of 8.4. Numerically, a pH of 7.0 is considered exactly neutral, below is acid, above is alkaline; hence, procaine borate is distinctly alkaline. Further experiments proved that the toxicity was due to the procaine base, the acid used to make the soluble salt having nothing to do with toxic action. There was also a relation between effectiveness and pH; the more alkaline the solution the greater was the effectiveness. If procaine hydrochloride was dissolved in a weak alkaline solution so as to have a pH of 7.3 to 8.3, it was found to be from ten to fifteen times as effective on goldfish as the original (more acid) solutions.

It may be explained that procaine is an insoluble alkaloidal base, and in order to make a soluble salt, hydrochloric acid is used, thus giving procaine hydrochloride, or boric acid may be used, giving procaine borate. An aqueous solution of procaine hydrochloride is acid in reaction with a pH of about 5.5; while procaine borate in aqueous solution is alkaline with a pH of 8.4. This wide variation in pH accounts for the great difference in the efficiency of the two salts.

Epinephrine must be used in local anesthetic solutions to retard absorption, thereby holding the solution within the field of operation. Epinephrine is a powerful drug and its rapid absorption may cause a rise of blood pressure with acceleration of the heart. Because of these physiologic effects, the dosage of epinephrine should always be kept at a minimum. Another important reason for using small amounts is that epinephrine is combined with hydrochloric acid, making epinephrine hydrochloride, the pH of which is about 3.3. This lowers the pH of the anesthetic solution which is undesirable. Experience with alkaline local anesthetic solutions reveals them to be much less toxic.

To make the alkaline vehicle, one gram of sodium carbonate is added to one litre of distilled water and sterilized in a cotton stoppered flask by boiling. The amount necessary for a patient is poured into a cup and the required number of T tablets of procaine hydrochloride and epinephrine added. This gives a solution with a pH of 7.8 slightly more alkaline than the blood, and produces a rapid anesthesia with virtually no postinjection disturbances. Injection of a solution of sodium carbonate alone is extremely irritating. When it is combined with the above tablet this chemical reaction occurs: $Na_2\ CO_3 + 2\ P.HCL \rightarrow 2\ NaCL + 2\ P + H_2\ O + CO_2$, or in other words, the injected solution contains procaine base, procaine hydrochloride and sodium chloride. This solution must be made fresh for each patient since an alkaline procaine solution begins to oxidize within one hour after preparation.

Procaine borate with epinephrine needs only to be dissolved in distilled water and sterilized, yielding a solution with a pH slightly over 8.0. This is effective very rapidly, giving complete anesthesia in mandibular injections in one to two minutes in many cases.

With such simple solutions available, and also simple methods of preparation, the many complex indefinite solutions on the market should be abandoned, and be thus assured of abusing the tissues of patients to a minimum.

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# BOOK REVIEWS

*Histo-pathology of the Teeth and their Surrounding Structures.* By Rudolf Kronfeld, M. D., Professor of Special Histopathology, Director of Department of Research, Chicago College of Dental Surgery, Dental Department of Loyola University, Chicago, Illinois; Former Assistant at the Dental Institute of the University of Vienna, Austria. Illustrated with 385 engravings, 479 pages.

Kronfeld's "Histo-pathology of the Teeth and their Surrounding Structures" was published in February of this year, and it is not extravagant to say that its appearance was a real event in the realm of dental literature. The book is of inestimable importance to all dentists, to the general practitioner as well as to the specialist, to the scientific-minded and to the clinician, because of the author's consistent reference to clinical observations and clinical problems in his discussions of pathological phenomena encountered in the mouth. His purpose has been to illustrate by means of human specimens the actual tissue changes that correspond to certain well defined clinical conditions. He emphasizes the use of human tissues, claiming that while animal sections may be very valuable in experimental work, still for the study of normal and pathological conditions of *human* teeth only perfect sections through normal and diseased *human* teeth and jaws can give the desired information. The assumption that the findings in the tissues of a sheep or a cat should be directly applicable to man is not substantiated and should be revised. The problem of obtaining human specimens is a difficult one especially when it is desirable to show the contiguous structures and attachments along with the teeth themselves. Many of Kronfeld's specimens are most exceptional; for example, we believe that photomicrographs of granulomas and cysts in situ within the jaw have perhaps never before been shown in a textbook of this kind and the importance and significance of these relations are well demonstrated by the proximity to the antrum of some of the cysts sectioned. Impacted teeth and root fragments are also shown in situ.

The chapter on tissue changes following root canal therapy is altogether new and those on root resorptions, on the biology of cementum and on the histology of edentulous jaws are largely so.

Kronfeld's book has already been reviewed in a general way (Jour. A. D. A. March, 1933 and Dental Cosmos, July, 1933) so we shall attempt to confine ourselves as closely as possible to the chapters dealing with Periodontology. The author has wisely prefaced this portion of his book with an excellent chapter on the histology of the epithelial attachment and gingival crevice, in which of course he adheres closely to the newer conception of histology advanced by the Vienna school.

In his classification of periodontal diseases also he follows Gottlieb, with some minor modifications which help correlate the clinical manifestations with the pathological picture. The resulting table, based thus on both clinical and laboratory observations makes a clear distinction between two main types of periodontal disease—a local form beginning with a marginal gingivitis, and a systemic form beginning with bone atrophy. The author has inserted, in parenthesis, the nomenclature suggested by the Federation Dentaire Internationale, but he is convinced "that any nomenclature is useless without a clear understanding of the pathological tissue changes associated with the various clinical forms of pyorrhea" and states further that "once the etiological factors are clear in the minds of both clinician and laboratory investigator it will be easy to find simple terms to serve as a means of mutual understanding". His table gives in a most condensed form Kronfeld's entire conception of periodontal disease, the clinical manifestations, etiology, pathology, and therapy of all the types, each of which subjects is elaborated in the succeeding pages. It has been established that the great majority of cases of pyorrhea are of the local type, Gottlieb's "Schmutzpyorrhea" and a practical suggestion is that a pyorrhea

diagnosis should only be made in a clean mouth.

The histo-pathology of gingival inflammation is presented so satisfactorily we should like to quote it in toto; we cannot too strongly emphasize its value. We read that the bottom of the crevice is the distinct line of demarcation that indicates how far rootwards calculus may extend and beyond which no deposits can be found (and no curettage is necessary). The benefit of brushing is scientifically demonstrated and two interesting photomicrographs show the contrast in gingival tissues and gingival crevice on the labial, brushed, and on the lingual, unbrushed, surface of two lower anterior teeth in the same mouth. On the labial, depth of crevice, crevice epithelium and all the components of the picture are normal; on the lingual, we see a deposit of calculus, the gingival crest is covered by desquamated epithelial cells, the crevice epithelium is broken, inflammatory cells are being discharged into the crevice and inflammatory changes are general in the soft tissues. Orban has recently shown that normal human gingiva under normal conditions and under proper function and brushing has a keratinized or hornified surface the presence of which is essential if a healthy epithelium is to be secured and maintained. Orban's slides were from tissues taken by biopsy and the importance of his findings can hardly be overestimated especially the fact that the unfavorable "unkeratinized" condition in the one is transformable by proper care into that shown in the other.

Of the healing of the deep pockets of "paradental pyorrhea" the author states that it must be understood that there is no likelihood of repair in the sense of the tissues reattaching to the root, the term healing meaning that the destructive process is arrested at a given level, not a regeneration to the status quo ante. "The denuded root surface inside the pocket represents a necrotic, macerated and infected hard tissue surface; under these circumstances, deposition of new layers of cementum, without which a functional reattachment is impossible, is unlikely and up to this time has not been demonstrated. The question of whether cementum or dentin is exposed in the crevice is without significance. It is known that new cementum may be deposited indiscriminately upon both cementum and dentin, for instance, following root fracture or root resorption. However, in these cases, the surface of dentin and cementum had never been denuded; whereas, in case of a paradental pocket, the dental hard substances have been exposed for months or years to infection and maceration."

Bacteria are secondary to other factors since they disappear promptly when the culture medium presented in any deep pocket is eliminated. . . . . The admittedly complex problem of alveolar atrophy is treated with enlightening detail under the following headings: 1. Physiological Atrophy of the alveolar margin as part of the general involution with advancing age. 2. Alveolar Atrophy due to excessive occlusal stress. 3. Alveolar Atrophy due to inflammatory processes in the gingival tissues. 4. Alveolar Atrophy as the expression of a systemic disturbance (diffuse atrophy). While the prognosis in cases of atrophy of the second and third classes may be considered favorable when discovered in time and when the causes are removed, that of the fourth type, the diffuse atrophy, of which exact etiology is not known, presents a more difficult problem and we are warned against the acceptance of unsubstantiated statements and conclusions on this subject. Lesions in the teeth and bone produced experimentally in animals by dietary means resemble only slightly conditions found in man,—the diet is utterly different and it is pointed out that in some of the most-cited experiments the bone changes produced in the animals were not confined to the jaw bone but were general all over the skeleton. If, however, the case history reveals distinct dietary or metabolic deficiency its correction will bring about a systemic improvement usually beneficial to the dental condition as well. The occlusal trauma, though secondary in these cases, must be kept under observation and relieved as it develops.

As is to be expected, traumatic occlusion is handled with scientific restraint. Two chapters treat of the influence of function —normal and excessive—on teeth and surrounding structures. Another correlated chapter, which contains much material that is wholly new in American dental literature deals with tissue changes in ortho-

dontia. The line of demarcation between physiological stress and pathological stress is difficult to establish clinically but under the microscope minute hemorrhages can be demonstrated in the periodontal membrane and sometimes small resorptions on the root which heal readily when the trauma is not excessive or is relieved within a reasonable time. "Whenever resorption of the root takes place the periodontal fibers lose their attachment to the involved area of the root surface; the corresponding alveolar bone will also be resorbed due to the loss of functional stimuli. The periodontal membrane thus becomes wider in the damaged area. When repair takes place, new cementum in which new fibers are embedded is deposited on the resorbed root surface; functional stimuli are again transmitted to the bone, which is regenerated until the original width of the functioning periodontal membrane is restored". This is functional repair. Lateral stresses are known to be more conducive to injury than axial stress and this phenomenon is explained by means of diagrams showing the mechanism of the tipping movement and by photomicrographs showing what changes are actually taking place. Whether teeth loosen under such stresses depends on individual tissue resistance and reaction. An especially significant photomicrograph is that of a tooth under excessive tipping stress in which extensive resorption of the alveolar bone has occurred, the lost bone having been replaced by fibrous connective tissue which is attached to the greater part of the root. The radiogram shows loss of alveolar bone to the very apex. This case is described in detail as typical of those in which an inaccurate diagnosis is made based solely on radiographic interpretation. If the pockets were explored the very considerable amount of soft tissue attachment (shown in the histological slide) would be discovered and the prognosis would be quite different, there being always possibility of regeneration of alveolar bone where there has been no detachment of the soft tissues.

The chapter on influence of function on the various tissues which make up the dental mechanism is original and most interesting. Excellent photo-micrographs show rarefied bone surrounding non-functioning teeth, and denser bone under functional stimulus. We see narrow periodontal membrane with few fiber bundles in non-functioning teeth, and broad membrane with dense regular fiber bundles under wholesome function. The deposition of new layers of cementum is apparently inhibited rather than stimulated by heavy function.

"Teeth without antagonists' impacted and erupting teeth, seem to have an inherent tendency to move occlusally. Occlusal displacement causes widening of the periodontal space—which is then reduced by compensatory deposits of cementum. After eruption when the tooth has reached its normal occlusion the periodontal space widens, well-defined fiber bundles develop between root and bone and the supporting bone becomes reinforced to withstand functional stress." Similarly in orthodontia cases where impacted teeth are moved, there is rearrangement of the blood vessels and nerves, transformation of narrow periodontal membrane into a wide fibrous one and development of a functioning alveolus.

It is with regret that we cannot go fully into the chapter on orthodontia in which Kronfeld summarizes the knowledge gained from the experiments of Oppenheim, Johnson, Appleton, Rittershofer and Schwarz. In fact there is not a chapter in the whole volume which does not merit careful perusal.

The author is to be congratulated on his achievement, and the dental profession scarcely less so for the opportunity afforded by his concise, clear and convincing presentation of subject matter on which the profession has felt the need for clarification.

Celia Rich, D.D.S.

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*The Status of Dental Journalism in the United States.* (Report of the Commission on Journalism of the American College of Dentists).

A complete survey of dental journalism, containing a history; a study of trade-house relationships and influences; nineteen tables of detailed data and statistics; an analysis of the conditions in dental journalism; conclusions and recommendations of the Commission and constructive critical comment regarding each of the important dental periodicals; and a bibliography on dental journalism from 1839 to 1931.

A copy of this 238 page work should be

in every dental and medical library, and should be read by every dentist with sufficient pride in his profession to wish to see its journalism elevated to a dignified status.

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*The New Dentistry—A Phase of Preventive Medicine.* By Leroy M. S. Miner, D. M. D., M. D., D. Sc. Dean of Harvard Dental School. pp. 210.

Popularly written, but valuable and authoritative.

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*Diet and the Teeth. An Experimental Study. Part III. The Effect of Diet on Dental Structure and Disease in Man.* By May Mellanby. 180 pp. of text. In addition there are 47 plates of most beautiful and instructive illustrations. Reviewed in the next issue of this Journal.

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# Apes, Men and Teeth

Perhaps no outstanding general scientist has so forcefully presented the importance of teeth as Dr. Ernest A. Hooton, Professor of Anthropology at Harvard University in an article entitled "Apes, Men and Teeth" in the January issue of the *Scientific Monthly*. Dr. Hooton supplies the evidence that life on earth has risen and fallen in its evolution more directly through the teeth than any other part of the anatomy. In fact he predicts the extinction of the human race unless we find some way of conserving the teeth. We recommend this fascinating article to every dentist and especially to orthodontists, who may find it of particular interest. The following are extracts from the article.

*U. G. Rickert*, D. D. S., Chairman of Committee on Coöperation, American Association of Dental Editors.

"Teeth tell the tale of human evolution better than any other bodily structure. No, I have not forgotten brains. But we do not understand the evolution of the human brain. Our brains are apparently much too big for the use we make of them. Certainly, neither we nor our immediate or remote ancestors have taken thought sufficiently to add many cubic centimeters to our cranial capacities. Yet here we are—all decked out in our 7¼ hats—and no place to go. We have less reason to be proud of our brains than to be ashamed of our teeth.

"Teeth seem to be a distasteful subject for popular consideration. They are unpleasantly reminiscent of the apprehensive minutes spent in the dental waiting room with the futile distraction of back numbers of cheery periodicals—and other far worse experiences. Yet our teeth have had an illustrious past; they have a serviceable present, and with due conservation they will continue to perform an indispensable function in the future of man. But if the human dentition breaks down, it will carry with it in its fall the human species.

"You must know the history of the teeth in order to appreciate them, and because this history epitomizes human evolution. The control of future human evolution is now in man's own hands—or perhaps rather in his teeth.

## *Short History of Teeth*

"Teeth are the most nearly imperishable relics of the vertebrate body. If an animal's teeth last until death, they will continue to defy the destructive action of time and the elements—sometimes for millions of years. After death the soft parts of the animal body decay rapidly. The bones are much tougher. But bones are frequently crushed into dust or dissolved by the action of chemicals in the earth. Furthermore, they are often and perhaps usually devoured by carnivorous animals for which they are a 'bonne bouche'. But teeth are singularly unpalatable and indigestible morsels. No animal eats them or, if it does, manages to digest them. They remain as monuments of extinct species. This is peculiarly fortunate for the student of evolution, since the teeth are not only the most durable, but also the most instructive of bodily parts.

## *How to Stop Dental Degeneration*

"In my opinion there is one and only one course of action which will check the increase of dental disease and degeneration which may ultimately cause the extinction of the human species. This is to elevate the

dental profession to a plane upon which it can command the services of our best research minds to study the causes and seek for the cure of these dental evils. Such an improvement of the quality of the dental profession is an indispensable prerequisite for the attacking of these tremendous pathological and evolutionary problems. No effective measures of public education in care of the teeth can be taken until dental practitioners cease to be *tinkers* and learn to be scientists. In making such a statement I by no means wish to belittle the tremendous progress in the field of dentistry which has already been made.

"As a matter of fact, if I were asked in what occupations the United States indubitably leads the world, I should reply without hesitation "Dentistry and *plumbing*." American dentists have reached a pitch of mechanical skill which is equal to that of American surgeons. But *carpentry* is not enough. Stopping teeth does not stop tooth decay. In the dental profession today are many brilliant scientific minds and many practitioners of consummate skill whose aims are humanitarian rather than pecuniary, but there are too few of such men and they have been insufficiently trained. (Italicized by editor.)

"The dental profession has been for too long a time a neglected and disowned orphan child of medicine and surgery. While millions have been lavished upon medical schools and hospitals, and upon medical and surgical research, almost nothing has been allotted for these purposes to dentistry. Our schools of dentistry have been forced to struggle along without endowments; their teaching staffs have consisted almost entirely of devoted but unpaid men who give part of their time to teaching, but have to make their living in practice."

[Dentists operate in a dimly lighted cavity, usually by reflected light on peculiarly sensitive tissue and with the patient seldom anaesthetized. They perform the most delicate and intricate operations requiring judgment as well as skill, without the glamour attendant upon general surgical operations. The success or failure of dentists' services may be readily seen by the next dentist or Roentgenologist the patient visits. The dentist has spent thousands of dollars and years of college preparation to equip himself for the practice of dentistry. Why then the *continued* association in literature of such terms as *tinkers, plumbing* and *carpentry* with one of the dignified and highly specialized health service groups?—Ed.]

THE Journal of Periodontology is lovingly dedicated to the memory of Doctor Gillette Hayden. Her selfless devotion and untiring efforts in behalf of periodontia and the American Academy of Periodontology, have served as an inspiration to her close associates which can only be consummated by carrying onward the work for which she spent her life.

THE JOURNAL OF PERIODONTOLOGY

*Published twice yearly—January and July—by*

*The* AMERICAN ACADEMY OF PERIODONTOLOGY

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*Publication and Editorial Office:* 555 W. MAPLE AVE., BIRMINGHAM, MICHIGAN

Single copies of this number may be obtained from
Publication Office at $1.00 each
1934 Subscription Price $2.00
All foreign subscriptions to be paid in United States money

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